

**RELATIONSHIP BETWEEN DAYTIME NAPPING AND CARDIOVASCULAR RISK  
FACTORS IN HEALTHY BLACK AND WHITE ADOLESCENTS**

by

**Karen P. Jakubowski**

B.S., B.A., University of Pittsburgh, 2011

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This thesis was presented

by

Karen P. Jakubowski

It was defended on

September 11, 2014

and approved by

Martica Hall, Ph.D., Professor of Psychiatry

Anna Marsland, Ph.D., Associate Professor of Psychology

Thesis Advisor: Karen Matthews, Ph.D., Distinguished Professor of Psychiatry

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# RELATIONSHIP BETWEEN DAYTIME NAPPING AND CARDIOVASCULAR RISK FACTORS IN HEALTHY BLACK AND WHITE ADOLESCENTS

Karen P. Jakubowski, M.S.

University of Pittsburgh, 2014

**Purpose:** Short nocturnal sleep leads to more napping in adolescents, and more napping leads to short nocturnal sleep. Short nocturnal sleep has been associated with elevated cardiovascular (CV) risk factors in adolescents in some studies. However, it is unclear if daytime napping is also associated with increased CV risk factors. We investigated whether napping (measured by actigraphy and daily diary measures) was related to elevated CV risk factors, and explored potential moderators of the napping – CV risk relationship: physical activity, sedentary behaviors, and interpersonal conflict.

**Methods:** Participants were 234 healthy high school students (mean age=15.7, 56% black, 53% female). Nocturnal sleep and daytime napping were assessed with both actigraphy and daily diaries across one week. Physical activity and interpersonal conflict were measured by daily diary and questionnaire; sedentary behaviors were measured with questionnaire only. CV risk factors included: sex-standardized waist circumference (WC), insulin resistance (HOMA-IR), high sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and 24-hour average systolic blood pressure (24-hour SBP). Linear regressions were used, adjusting for age, sex, race, average nocturnal sleep across the study period, and BMI percentile.

**Results:** More days napped by actigraphy was related to elevated IL-6 [B(SE)=.49(.21),  $p < .05$ ] but *lower* 24-hour SBP [B(SE)=-5.29(2.64),  $p < .05$ ]. Napping was not associated with WC, HOMA-IR, or hs-CRP. The relationship between average minutes napped by actigraphy and 24-

hour SBP varied as a function of life events conflict ( $b = -.76$ ,  $SE = .31$ ,  $p = .015$ ). At high levels of conflict, napping was associated with lower levels of 24-hour SBP, while at low or average levels of conflict, there was no effect of napping on 24-hour SBP. Neither physical activity nor sedentary behaviors emerged as moderators.

**Conclusions:** No consistent association of napping with CV risk factors was observed. However, actigraphy-derived napping was associated positively with circulating IL-6, a proinflammatory cytokine that is known to impact central inflammatory processes that relate to sleep regulation. Further examination of the direction of this effect is warranted. Results may differ in other age groups who are not as sleep deprived as is the case among adolescents.

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## **1.0 INTRODUCTION**

An expansive literature has described the discrepancy between the recommended amount of nocturnal sleep for adolescents (approximately 9 hours per night) and the evidence that almost 80% of adolescents fail to achieve that amount (National Sleep Foundation, 2006). A number of biological, behavioral, social, and school-related explanations for this sleep loss have been postulated, with associated deficits in academic and emotional functioning, as well as adverse physical health and safety outcomes. Considering physical health, evidence indicates associations between short nocturnal sleep and elevated ambulatory blood pressure (Mezick, Hall, & Matthews, 2012), insulin resistance (Matthews, Dahl, Owens, Lee, & Hall, 2012; Javaheri, Storfer-Isser, Rosen, & Redline, 2011), body mass index and waist-hip ratio (Garaulet et al., 2011), and inflammation (Hall, Lee, & Matthews, 2014; Martinez-Gomez et al., 2011).

Given the deficits in adolescent nocturnal sleep, it is not surprising that adolescents report napping a lot. According to a 2011 National Sleep Foundation (NSF) poll, approximately 53% of 13-18 year olds reported napping on weekdays (National Sleep Foundation, 2011). Yet the impact of napping on nocturnal sleep and biological outcomes has been relatively unexplored. It is possible that napping may have an important restorative function by allowing chronically sleep-deprived adolescents to catch up on sleep, thus serving as a positive health behavior; or perhaps napping is better viewed as a marker of sleep deprivation, one that causes further dysregulation of the sleep-wake cycle. Given the paucity of evidence on napping, it is important to understand its impact on adolescent health, independent of the impact of nocturnal sleep.

To date, no studies in adolescents have examined the effect of napping separately from total sleep time on biological outcomes that may indicate emerging cardiovascular risk markers. Consequently, the present study tested hypotheses that napping, independent of nocturnal sleep duration and BMI percentile, was related to the following cardiovascular (CV) risk factors: (1) *sex-standardized waist circumference*; (2) *insulin resistance* (homeostatic model assessment of insulin resistance; HOMA-IR); (3) *high sensitivity C-reactive protein* (hs-CRP); (4) *interleukin-6* (IL-6); and (5) *24-hour average systolic blood pressure* (24-hour SBP). Napping was measured by actigraphy and diary and consisted of proportion of days napped and average minutes napped across the seven-day study period. Exploratory analyses aimed to identify potential moderators of the relationship between napping and CV risk factors: (1) daily diary and questionnaire measures of *physical activity*, (2) questionnaire measure of *sedentary behaviors*, and (3) daily diary and questionnaire measures of *interpersonal conflict*. These moderators were chosen because they have been associated with short nocturnal sleep and may exacerbate the relationship between napping and CV risk.

## **2.0 WHY DO CV RISK FACTORS MATTER IN ADOLESCENTS?**

Evidence suggests that CV risk factors are increasing in prevalence during childhood and adolescence, particularly rates of pre-diabetes and hyperinsulinemia (Li, Ford, Zhao, & Mokdad, 2009), systolic and diastolic blood pressure (Muntner, He, Cutler, Wildman, & Whelton, 2004), and obesity (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). These elevated risk factors, particularly blood pressure and body mass index (BMI), may track into adulthood to predict future CV risk (Hartiala et al., 2012; Rademacher et al., 2009; Li et al., 2003; Raitakari et al., 2003). Indeed, these findings highlight the importance of investigating CV risk at younger ages.

### **3.0 DOES SLEEP RELATE TO CV RISK FACTORS IN ADULTS?**

#### **3.1 METABOLIC RISK AND CARDIOVASCULAR DISEASE (CVD).**

Both short and long self-reported sleep has been associated with increased risk for the development of type 2 diabetes (Cappuccio, D'Elia, Strazzullo, & Miller, 2010), morbidity and mortality from coronary heart disease and stroke (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011), as well as all-cause mortality (Cappuccio, D'Elia, Strazzullo, & Miller, 2011; Gallicchio & Kalesan, 2009). Short sleep is related to risk for metabolic syndrome (Xi, He, Zhang, Xue, & Zhou, 2013), while long sleep is related to total CVD events (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011).

#### **3.2 OBESITY**

A meta-analysis by Cappuccio et al. (2008) found a negative association between hours of sleep (for most studies,  $\leq 5$  hours/night) and BMI. Although the majority of studies focused on adults, some included adolescents as young as 15. Patel and Hu (2008) have suggested the evidence concerning the association between short sleep and obesity in adults is mixed, in part due to some studies finding a U-shaped relationship. Paragraph. The table below is included so that there is an item in the sample List of Tables.

### **3.3 BLOOD PRESSURE**

A number of meta-analyses have indicated a relationship between self-reported short sleep and increased risk for hypertension (Guo et al., 2013; Meng, Zheng, & Hui, 2013; Palagini et al., 2013; Wang, Xi, Liu, Zhang, & Fu, 2012). Although the evidence is more mixed for the association between long sleep duration and hypertension risk, meta-analyses by Guo et al. (2013) and Wang et al. (2012) have provided support for this association.

### **3.4 INFLAMMATORY MARKERS**

CRP is an acute phase reactant marker of inflammation synthesized in hepatocytes (de Ferranti & Rifai, 2002). High-sensitivity CRP (hs-CRP) has been associated with atherosclerosis (Blake & Ridker, 2002) and has prospectively predicted CV events in both healthy subjects and those with coronary disease (Danesh et al., 2000). IL-6 is a proinflammatory cytokine that is produced by activated immune cells, as well as by a number of other cell subtypes, including adipocytes and myocytes. IL-6 plays a role in coordinating local and systemic inflammatory responses and also communicates with the central nervous system to induce behavioral changes that include sleep dysregulation and the experience of fatigue (Cho, Bower, Kiefe, Seeman, & Irwin, 2012); IL-6 also stimulates CRP production from the liver (Buchan, Young, Boddy, Malina, & Baker, 2013).

There is mixed support for the relationship between sleep duration and inflammatory markers in adults. Evidence indicates a U-shaped association between sleep duration and CRP (Grandner et al., 2013; Miller et al., 2009), although Miller and colleagues found this association in women only. Also in women, short sleep measured by polysomnography (PSG) was related to



increased IL-6 (Miller et al., 2009) and CRP (Matthews et al., 2010). In contrast, Taheri and colleagues (2007) found no association between PSG-measured sleep duration and CRP.

### **3.5 SUMMARY**

It is important to note that meta-analyses on adult sleep are somewhat limited by methodological differences across studies on definitions of “short” and “long” sleep. Yet the previously described results demonstrate relationships between self-reported short sleep (and less often, long sleep) and poorer CV health and all-cause mortality. These findings warrant the investigation of the relationship between sleep and CV risk factors in younger age groups, particularly adolescents, to determine how early these relationships may be observed.

#### **4.0 DOES DAYTIME NAPPING RELATE TO CV RISK FACTORS IN ADULTS?**

Evidence for the relationship between napping and CV risk has been mixed in epidemiological studies of adults. Although older adults may nap for different reasons than adolescents, (e.g., medical conditions, less structured time due to retirement), there are likely similarities as well (e.g., short nocturnal sleep, daytime sleepiness). Some studies suggest that napping is associated with worse CV outcomes, even in relatively healthy adults without a history of CVD, stroke, or cancer. Adults with a regular daytime napping habit had an elevated risk of cardiac events (Stang et al., 2012; Campos & Siles, 2000), CVD mortality (Tanabe et al., 2010) and mortality from all causes (Leng et al., 2014; Tanabe et al., 2010). In contrast, some epidemiological studies have demonstrated *protective* effects of napping. Brindle and Conklin (2012) found that healthy university students who obtained more daytime sleep demonstrated faster blood pressure recovery to a mental stress task, relative to students who obtained less daytime sleep. Naska and colleagues (2007) found an inverse relationship between napping and coronary mortality in healthy adults, although they did not adjust for nocturnal sleep duration.

## **5.0 DOES SLEEP RELATE TO CARDIOVASCULAR RISK IN ADOLESCENTS?**

### **5.1 OBESITY**

BMI is a risk factor that has been frequently studied in relation to sleep duration in children and adolescents. Meta-analyses have indicated a relationship between short sleep (usually self-reported) and increased BMI or risk of obesity in children and adolescents (Cappuccio et al., 2008; Chen, Beydoun, & Wang, 2007; Patel & Hu, 2008). Gauralet and colleagues (2011) found a relationship between self-reported short sleep (<8 hours) and both increased waist and hip circumferences and BMI in adolescents aged 12 to 17 years.

### **5.2 BLOOD PRESSURE**

Javaheri, Storfer-Isser, Rosen and Redline (2008) found that pre-hypertensive adolescents were likely to have low actigraphy-assessed sleep efficiency and short sleep duration; the pattern of results were consistent when sleep was measured by PSG. Mezick et al. (2012) reported that shorter actigraphy-assessed sleep duration across one week was related to higher 48-hour blood pressure and higher nighttime BP in white, but not black adolescents.

### 5.3 INSULIN RESISTANCE

The relationship between sleep and insulin resistance in adolescents has not been extensively studied, although results are mixed. Javaheri and colleagues (2011) found a U-shaped relationship between actigraphy-assessed sleep and HOMA in healthy adolescents, while Koren and colleagues (2011) reported a U-shaped relationship between PSG-measured sleep duration and hyperglycemia in obese youth. Evidence also suggests associations between *short* sleep only and insulin resistance, using sleep measured by actigraphy and daily diaries (Matthews et al., 2012), as well as PSG (Flint et al., 2007). Additionally, Spruyt, Molfese, & Gozal (2011) reported that short sleep was associated with altered insulin.

### 5.4 INFLAMMATION

Martinez-Gomez and colleagues (2011) found an inverse relationship between diary-reported sleep duration and CRP levels, but not IL-6. Hall, Lee, and Matthews (2014) found that shorter actigraphy-assessed weekday sleep duration predicted risk of inclusion in a high-risk CRP group (CRP values > 3), while Spruyt et al. (2011) reported that short sleep duration was associated with higher CRP levels in children. Overall, there is still little evidence for the relationship between inflammatory markers, particularly IL-6, and sleep in adolescents.

## **5.5 SUMMARY**

There are demonstrated relationships between sleep and adverse CV risk outcomes in both adults and adolescents. However, the evidence in adolescents is still limited and excludes analysis of daytime napping. It is imperative to fill these gaps, specifically using both objective and self-report sleep measurements in relation to CV risk factors.

## **6.0 RELATIONSHIP BETWEEN SLEEP AND HEALTH BEHAVIORS/INTERPERSONAL CONFLICT IN ADOLESCENTS**

In addition to CV risk, short nocturnal sleep may be related to emotional dysregulation and poorer health behaviors, including decreased physical activity and increased sedentary behaviors. The role of napping in affecting these factors is not well understood. Understanding how nocturnal sleep is related to napping, and whether napping is associated with these health behaviors and conflict sets the stage for exploring if these behaviors further explicate the nature of the relationship between napping and CV risk.

### **6.1 HEALTH BEHAVIORS**

Few studies have examined the relationship between nocturnal sleep and physical activity in adolescents. Evidence suggests that shorter sleep is associated with less physical activity in adolescents (Countryman et al., 2013; Stea, Knutsen, & Torstveit, 2014), and importantly, decreased self-reported physical activity has been associated with increased risk for metabolic syndrome, insulin resistance, and elevated IL-6 and CRP (Countryman et al., 2013; Platat et al., 2006). Therefore, factors that contribute to reduced physical activity in adolescents may play a role in increased CV risk in this population.

Napping may both perpetuate a cycle of short nocturnal sleep *and* contribute to reduced physical activity among adolescents, although no studies have reported the relationship between napping and physical activity in adolescents. In healthy older adults, those who napped daily were less likely to report being moderately active/active relative to non-nappers (Leng et al., 2014), and were also less likely to report walking for exercise than non-nappers (Stone et al., 2009). It is likely we would see a similar pattern of reduced physical activity among adolescent nappers, given evidence of a bidirectional relationship between short nocturnal sleep and napping (Jakubowski, Hall, Lee, & Matthews, 2014), a cycle that may perpetuate decreased physical activity. Furthermore, data in adolescents indicates that less engagement in physical activity-related behaviors is associated with various health risk behaviors, such as cigarette smoking (Nelson & Gordon-Larsen, 2006). Thus the relationship between napping and CV risk would be even worse in adolescents who demonstrate low physical activity, which may be a marker of overall dysregulation of a number of health behaviors related to CV risk.

Sedentary behaviors independently predict risk for adverse cardiovascular and metabolic outcomes in both adults (Hamilton et al., 2008) and adolescents (Tremblay et al., 2011). Data from healthy older adults suggests that daily nappers were less likely to report being moderately active/active relative to non-nappers (Leng et al., 2014). Among adolescents, evidence indicates an association between short sleep and increased sedentary behaviors (Garaulet et al., 2011; Stea et al., 2014), as well as a bidirectional relationship between short sleep and napping (Jakubowski et al., 2014); thus it is plausible that napping perpetuates the relationship between short sleep and sedentary behaviors. Sedentary behaviors, particularly screen time, may exacerbate the expected negative effect of napping on CV risk, because higher engagement in screen time has been associated with various health risk behaviors in adolescents (Nelson & Gordon-Larsen, 2006).

Thus the napping – CV risk relationship would be even worse in adolescents who report increased engagement in sedentary behaviors, as this may be a marker of overall dysregulation of a variety of health behaviors related to CV risk. It is important to study the moderating role of sedentary behaviors separately from physical activity, as these behaviors are unique constructs with independent relationships with CV risk (e.g., Eisenmann, Bartee, Smith, Welk, & Fu, 2008).

## **6.2 INTERPERSONAL CONFLICT**

Short nocturnal sleep is associated with poorer emotion regulation. Evidence from experimental sleep restriction studies have demonstrated associations between sleep deprivation and increased reactivity to negative emotional stimuli (Franzen et al., 2009), increased affective instability (Yoo et al., 2007), and decreased ability to regulate emotions the next day, producing low frustration tolerance and irritability (Franzen et al., 2008). Thus under conditions of inadequate sleep, it is likely that individuals would demonstrate more difficulty managing interpersonal stressors. Considering that short nocturnal sleep is associated with more napping, and more napping leads to short nocturnal sleep in adolescents (Jakubowski et al., 2014), napping may perpetuate the relationship between short sleep and poorer emotion regulation. Thus the costs of napping on CV risk factors would be stronger in adolescents who are more vulnerable to the effects of short nocturnal sleep on emotion regulation, particularly those who report increased interpersonal conflict.



### **6.3 SUMMARY**

The relationship between napping and CV risk factors may be strengthened in the context of low physical activity, high sedentary behaviors, or high interpersonal conflict. Next, I will present evidence that suggests daytime napping is inversely related to nocturnal sleep; in consequence, adolescents who nap may display poorer CV risk factors.

## **7.0 RELATIONSHIP BETWEEN NOCTURNAL SLEEP AND DAYTIME NAPPING**

The previously reviewed literature indicates that short or inadequate nocturnal sleep in both adults and adolescents negatively impacts CV health, health behaviors, and mood. In order to understand how daytime napping impacts these factors, it is important to place napping in the context of nocturnal sleep, using the 2-process model of sleep regulation (Borbély, 1982).

### **7.1 NOCTURNAL SLEEP**

Borbély (1982) reported that sleep is regulated by two physiological processes, the circadian rhythm and the homeostatic sleep drive. Sleep drive, or our need for sleep, begins to increase as soon as one wakes up in the morning, and continues to increase until one goes to bed. Ideally, adolescents would go to bed at a time that would allow them to obtain the recommended nine hours of sleep. Unfortunately, evidence suggests many adolescents obtain fewer than eight hours per night (e.g., McKnight-Eily et al., 2011; Roberts, Roberts, & Xing, 2011; Roberts, Roberts, & Duong, 2009). A biological change during puberty involves a circadian delay, such that adolescents prefer later bedtimes and wake times, even when wake times are constrained by early school start times. More time is spent in social and activities and utilizing technology before bed, leading adolescents to prefer wake activities over sleep. Thus a combination of factors makes it difficult for adolescents to obtain adequate nocturnal sleep.

## 7.2 NAPPING

In an adolescent who has obtained insufficient nocturnal sleep, he or she may experience feelings of sleepiness during the day and take a nap. Although this may alleviate feelings of sleepiness, napping negatively alters the sleep drive, decreasing sleep pressure, and causing adolescents to go to bed even later and sleep even shorter that night. Evidence from Project Pressure suggests that napping may interfere with adolescents' nocturnal sleep and cause further dysregulation in their sleep patterns, as would be expected from the two-process model of sleep. Shorter actigraphy-assessed nocturnal sleep duration predicted more next-day napping (by actigraphy and diary measures); while more napping (by actigraphy and diary) also predicted shorter same-day nocturnal sleep duration and later bedtimes (Jakubowski et al., 2014). These results supported previous work from Fischer, Nagai, and Teixeira (2008), who found that more actigraphy-assessed minutes napped on school days predicted shorter same-night sleep duration in a sample of Brazilian adolescents. Thus evidence suggests that napping interferes with nocturnal sleep in adolescents, and may not be a health-promoting behavior in this group.

## 8.0 SCOPE OF THE PROPOSED INVESTIGATION

Evidence in adults suggests that napping may have adverse effects on CV health. Given the relationship between short nocturnal sleep and CV risk in adolescents, and the demonstrated relationship between short sleep and daytime napping, it is possible that napping may be associated with poorer CV health in adolescents as well. Based on the evidence that daytime napping predicted later bedtimes in adolescents, in combination with the fixed early school schedule they must follow, the present study posited that napping would be related to elevated CV risk factors, as napping continues the cycle of shortened sleep. In order to understand the impact of napping on CV risk, it is important to look at napping separately from nocturnal sleep; as a result all analyses tested for the main effect of napping on CV risk.

Thus the primary aim of the proposed study was to test whether actigraphy- and diary-assessed daytime napping (proportion of days napped and average minutes napped across the study period) was associated with elevations in CV risk factors with known relationships to both nocturnal sleep and CV health: (1) sex-standardized waist circumference; (2) HOMA-IR; (3) hs-CRP; (4) IL-6; and (5) 24-hour SBP. It was hypothesized that napping would be related to elevated CV risk (*Hypothesis 1*). Furthermore, it was hypothesized that napping would be related to CV risk independent of average nocturnal sleep throughout the weeklong study period (*Hypothesis 2*), and both average nocturnal sleep and BMI percentile, a CV risk factor that is highly correlated with waist circumference and other CV risk factors (*Hypothesis 3*).

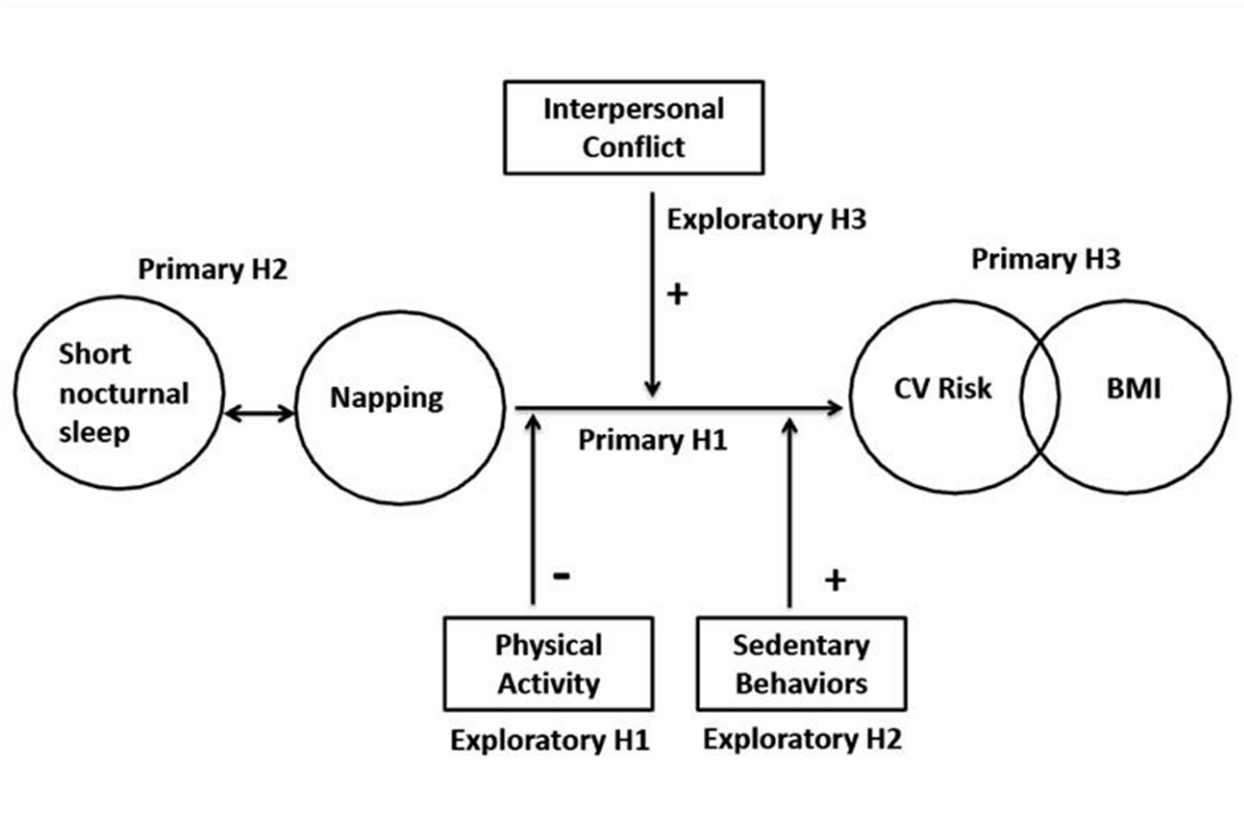
Three exploratory aims investigated potential moderators of the relationship between napping and CV risk. First, I aimed to test whether physical activity moderated the relationship between napping and CV risk (**Exploratory Aim 1**). Evidence in adults indicates that napping is related to poorer CV health (e.g., Stang et al., 2012), and that nappers report less physical activity (Leng et al., 2014). Among adolescents, we would expect decreased physical activity to worsen the relationship between napping and CV risk factors because decreased physical activity has been associated with both worse CV health (Countryman et al., 2013; Platat et al., 2006) and other risky health behaviors in adolescents (Nelson, Gordon-Larsen, 2006). Thus adolescents who nap *and* are less physically active are likely experiencing dysregulation across both sleep and health behavior domains, which may interact to produce elevated CV risk. We hypothesized that decreased engagement in physical activity would exacerbate the negative effect of napping on CV risk (*Exploratory Hypothesis 1*). To address this aim, I used both daily diary- and questionnaire-measured physical activity.

Second, I aimed to test whether sedentary behaviors moderated the relationship between napping and CV risk (**Exploratory Aim 2**). Evidence in adults indicates that napping is related to poorer CV health (e.g., Stang et al., 2012), and that nappers are more likely to report increased inactivity relative to non-nappers (Leng et al., 2014). Among adolescents, we would expect increased sedentary behaviors to worsen the relationship between napping and CV risk factors because sedentary behaviors have been associated with both worse CV health (Tremblay et al., 2011) and other risky health behaviors in adolescents (Nelson, Gordon-Larsen, 2006). Thus adolescents who nap *and* are more sedentary may be dysregulated across both sleep and health behaviors domains, which may interact to produce elevated CV risk. Thus we hypothesized that increased engagement in sedentary behaviors would exacerbate the negative effect of napping on

CV risk (*Exploratory Hypothesis 2*). To address this aim, I used questionnaire report of hours engaged in sedentary behaviors on an average school day.

Third, I aimed to test whether interpersonal conflict moderated the relationship between napping and CV risk (**Exploratory Aim 3**). Evidence suggests short nocturnal sleep is associated with poorer emotion regulation. Furthermore, short nocturnal sleep is bidirectionally associated with napping in adolescents (Jakubowski et al., 2014); as a result, napping may perpetuate the relationship between short sleep and poorer emotion regulation. Adolescent nappers who were more vulnerable to the effects of short nocturnal sleep on emotion regulation *and* who reported increased interpersonal conflict were predicted to show a stronger relationship between napping and CV risk factors. Thus it was hypothesized that the effect of napping on CV risk would be stronger in adolescents reporting increased interpersonal conflict (*Exploratory Hypothesis 3*). To address this aim, I used both daily diary- and questionnaire-measured interpersonal conflict. Figure 1 depicts the overall study model, including primary and secondary aims and hypotheses.

**Figure 1.** Overall Study Model



## **9.0 METHODS**

### **9.1 PARTICIPANTS**

Participants were 250 (47% male and 56% black) healthy adolescents between the ages of 14 and 19 ( $M = 15.7$  years) enrolled in Project Pressure, a project designed to measure risk factors for cardiovascular disease in adolescents. Participants attended a socioeconomically and ethnically diverse public high school near Pittsburgh, and were recruited from school health classes. Participants, and in the case of students under the age of 18, a parent or legal guardian, provided written informed consent prior to any research procedures. Sixteen students who were screened were ineligible to participate due to taking medication that could affect study variables, and seven students who signed consent did not actively enroll in the study.

Given evidence that five or more nights of adequate data are necessary to provide reliable estimates of actigraph-assessed sleep in adolescents (Acebo et al., 1999), the minimum criteria for inclusion into the analytic sample was at least five nights of actigraphy and diary data and also at least one CV risk factor. Sixteen participants were excluded from all analyses because of malfunctioning actigraph ( $N=1$ ), less than 5 days of actigraphy and diary data ( $N=13$ ), or BMI values that fell more than 4 standard deviations from the mean ( $N=2$ ). These exclusions resulted in a final sample of 234 adolescents, although sample sizes differed slightly across analyses



owing to missing CV risk factors or questionnaire data for some participants; see Table 1 for characteristics of the full analytic sample.

## **9.2 MEASURES**

### **9.2.1 Demographic information**

Adolescents self-reported age, gender, and race. Family socioeconomic status was determined from parental/caregiver report on the Hollingshead Four Factor Index (Hollingshead, 1975). This scale measures socioeconomic status by coding paternal and maternal years of education and highest attained degree, as well as current occupation for both parents (if contributing to the household income) to yield an overall score.

### **9.2.2 Pubertal status**

Adolescents completed the Peterson pubertal development scale (Peterson, Crockett, Richards, & Boxer, 1988), a 5-item self-report questionnaire designed to assess pubertal development based on self-reported growth in height body and skin changes, as well as gender-specific pubertal development changes. For analytical purposes, 0 = beginning/middle pubertal status, 1 = advanced/post pubertal status.

### 9.2.3 Actigraphy

The Mini-mitter actiwatch model AW-16 (Phillips Respironics, Bend, OR) was used to collect sleep/wake activity continuously over seven days and nights. The watches were configured to collect data over a 1-minute epoch. The medium threshold (default) was selected to detect nocturnal sleep periods of at least 3 hours in duration based upon sleep onset and offset using the 10-minute criteria of quiescence. Sleep periods occurring within 30 minutes of the major nocturnal sleep interval (either 30 minutes prior to sleeping or after waking) that were at least 15 minutes in duration were combined with the major sleep interval (i.e., if a 6-hour sleep interval was detected from 12 a.m. – 6 a.m., and a 20-minute sleep interval was detected beginning at 11:30 p.m., the 20-minute interval was combined with the major sleep interval. The new major rest interval would become 11:30 p.m. – 6 a.m.). All sleep variables were calculated within these set rest periods. Data were downloaded into the Actiware software program (version 5.57) for processing and analysis. Sleep duration was calculated as the number of hours between initial sleep onset and final sleep offset, excluding periods of wakefulness throughout the night.

Periods of napping were required to meet the ten-minute criterion of quiescence as described for nocturnal sleep; nap periods were measured in minutes. Napping variables included the following: proportion of days napped across the study period (i.e., total number of days with at least one nap divided by number of days with good actigraphy data) and average minutes napped across the study period. The variable for average minutes napped across the study period was positively skewed, therefore natural log transformation will be used in analysis after adding 1. Although PSG is the “gold standard” of sleep measurement, the actiwatch has been used extensively in research, and has been validated against PSG (Tryon, 2004; Kushida et al., 2001).

#### **9.2.4 Daily diary**

Adolescents completed diaries on a handheld computer each morning after awakening and each night before going to bed during the seven-day study period. In the morning diary, adolescents reported time went to sleep and awoke, from which a diary-reported sleep duration variable was computed. In the nighttime diary, adolescents reported minutes napped, engagement in physical activity, and instances of interpersonal conflict from the day. As with actigraphy, diary-reported napping variables included: proportion of days napped across the study period (i.e., total number of days with at least one self-reported nap divided by number of days with diary data) and average minutes napped across the study period. Both diary nap variables were positively skewed and underwent square root transformation (i.e., proportion of nap days), or natural log transformation (i.e., average minutes napped) after adding 1.

##### **9.2.4.1 Physical activity**

In the daily diary, adolescents responded yes or no to engagement in physical activity that day (i.e., “Did you exercise during the course of the day?”). For analytical purposes, “yes” responses were summed across the seven-day study period and divided by the number of days with diary data to provide a proportion of their daily physical activity throughout the week.

##### **9.2.4.2 Interpersonal conflict**

Adolescents reported in the diary whether any of the following events had happened that day: arguments/tension with family, friends, or others (e.g., teacher, boss). For analysis, the number of days with any conflict events (0 = no conflict events reported that day, 1 = conflict event

reported that day) were summed across the study period and divided by the number of days with valid diary data to provide a proportion of days with any conflict.

The daily diary interpersonal conflict variable was correlated with self-reported measures of (1) trait negative affect using the Positive and Negative Affective Schedule (PANAS; Watson, Clark, & Tellegen, 1988), (2) negative/conflictual life events that occurred in the last year using the Life Events Questionnaire – Adolescent (Masten, Neemann, & Andenas, 1994), and (3) EMA-measured negative affect (PANAS ratings reported in the blood pressure diaries for two school days during the study period). Pearson correlations between the diary-reported interpersonal conflict measure and measures of daily negative affect, trait negative affect, and conflictual life events were low ( $r$ 's = 0.2 – 0.3) but significant ( $p$ 's < .01). Since these measures of negative affect and interpersonal conflict were all significantly related to the interpersonal conflict measure, there was evidence supporting the interpretation of the proposed measure.

### **9.2.5 Questionnaires**

Psychosocial questionnaires were completed via protected web access.

#### **9.2.5.1 CDC Youth Risk Behavior Surveillance System Survey**

The Youth Risk Behavior Surveillance System Survey (YRBSS; Centers for Disease Control and Prevention, 2007) was used to measure smoking, physical activity, and sedentary behaviors. To measure smoking, adolescents responded to the question “During the past 30 days, on how many days did you smoke cigarettes?” Responses were categorized as 0 = did not smoke cigarettes, 1 = smoked cigarettes on one day or more in the past 30 days. To measure physical activity,

participants reported the number of days during the past seven that they were physically active for at least 60 minutes. This variable was examined continuously (0 to 7 days).

Two items from the YRBSS were selected to measure sedentary behaviors. Participants reported (1) number of hours spent watching TV on an average school day, and (2) number of hours spent playing video/computer games or using a computer for something other than school work on an average school day. Responses were scored as follows: “0” = I do not watch TV on an average school day; “1” = Less than 1 hour per day; “2” = 1 hour per day; “3” = 2 hours per day; “4” = 3 hours per day; “5” = 4 hours per day; and “6” = 5 or more hours per day. Responses from the two questions were averaged to form a variable of average number of hours engaged in sedentary behaviors on a typical school day; this was examined continuously.

#### **9.2.5.2 The Life Events Questionnaire – Adolescent**

The Life Events Questionnaire – Adolescent (LEQ-A; Masten et al., 1994) was developed to understand the relationship between stressful life experiences and adolescent adjustment. Students were asked to indicate whether a particular life event occurred in the past 12 months. Interpersonal conflict life events that are endorsed will be summed to obtain a possible range from 0 to 5: many arguments between adults living in the house; many arguments with brothers and/or sisters; many arguments with parents; close friendship ended; stopped dating. Chronbach’s Alpha for conflict events = .58 (5 items), N=231.

#### **9.2.5.3 Center for Epidemiological Studies – Depression**

Depressive symptoms were assessed using the 20-item Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977). Participants rated the frequency of experiencing each symptom in the past week, ranging from 1 (less than one day) to 4 (five to seven days). A sample

item was “I was bothered by things that usually don’t bother me.” Higher scores indicated greater depressive symptoms. One question pertaining to sleep disturbance (“My sleep was restless”) was removed from the total depression score used in analyses. Cronbach’s alpha coefficient for the CES-D in this sample was 0.74.

## **9.2.6 CV risk factors**

### **9.2.6.1 Waist circumference**

Waist circumference was taken at the point of natural bend of the waist under clothing and after two forced exhalations. Waist circumference values were standardized within age and sex.

### **9.2.6.2 Insulin resistance**

Morning fasting blood samples were obtained and serum was separated by refrigerated centrifuge, then aliquoted and stored at -62°C until assay at the Heinz Lipid Laboratory at the University of Pittsburgh. Serum glucose was assessed by an enzymatic determination using coupled enzyme reactions (Bondar & Mead, 1974). Insulin was measured using an RIA procedure. The limit of sensitivity was 2uU/ml to 200uU/ml in a linear fashion. The HOMA-IR was calculated as the product of the fasting glucose and insulin divided by the constant 22.5 (Matthews et al., 1985). The intra- and inter-assay coefficients of variation were 1.8% and 10%, respectively. Due to skewness, HOMA-IR was log transformed after adding 1.

### **9.2.6.3 High sensitivity C-reactive protein**

High-sensitivity CRP (hs-CRP) was selected as a summary marker of systemic inflammation, as it has a long half-life and is detectable at low levels. CRP was measured turbidimetrically by

measuring increased absorbance when the CRP in the sample reacts with anti-CRP antibodies. The intra- and inter-assay coefficients of variation were 5.5% and 3.0%, respectively. Due to skewness, CRP was log transformed after adding 1.

#### **9.2.6.4 Interleukin-6**

Interleukin-6 (IL-6) was selected as a marker of CV risk, as it is known to mediate the amplification of pro-inflammatory signals within atherosclerotic plaque (Blake & Ridker, 2002). IL-6 was determined colorimetrically using a high-sensitivity enzyme-linked immunosorbent assay (ELISA). The intra- and inter-assay coefficients of variation were 6.6% and 4.9%, respectively. Due to positive skewness, IL-6 was log transformed after adding 1.

#### **9.2.6.5 Ambulatory blood pressure**

Ambulatory BP was measured using the Spacelabs model 90217 monitor (Issaquah, WA). The BP machines were worn for two school days and nights and were programmed to take BP readings every 30 minutes from 7 a.m. to 10 p.m. and hourly from 10 p.m. to 7 a.m. Participants were told to wear the monitor at all times except when bathing. Software programs matched the diary entries to the appropriate BP reading. BP readings classified as nighttime pressures were in the interval between when the participant reported trying to go to sleep and awakening in the morning. Data analysis involved 24-hour SBP, defined as the average of all valid readings obtained across the 2- to 3-day period.

### **9.3 PROCEDURE**

Parents of students expressing interest in the study were contacted for a screening telephone interview to determine if the parent was willing to have their child participate, and also to determine students' eligibility (i.e., confirm that the student was free of cardiovascular or kidney disease, not taking medications for emotional problems or diabetes, or medications known to affect the cardiovascular system or normal sleep). After obtaining informed consent from the parent/guardian and the student, the student was scheduled to complete a seven-day study protocol, involving a fasting blood draw, anthropometric measurements, and wearing the ambulatory blood pressure monitor. The blood draw was done in the morning by trained personnel after verifying that the student had been fasting for at least eight hours and was not taking medications for infectious disease on the day of the draw. Participants who reported being ill were rescheduled. The blood was drawn in the recumbent position using standard procedures. Participants wore an actigraph on their non-dominant wrist continuously for seven days and nights. They reported napping, physical activity, and interpersonal conflict on a handheld computer each evening, and also completed a battery of psychosocial questionnaires. After successful completion of the protocol participants were paid \$100. A follow-up report of the student's blood pressure, sleep, and lab results was sent to the student and the parent/guardian.

### **9.4 ANALYTIC PLAN**

Of the 234 adolescents who had adequate sleep and BMI data, 10 participants were additionally missing at least one CV risk factor outcome: one reported symptoms of acute infection at the



time of the blood draw (removed from CRP analyses), two had incomplete nocturnal BP data because of taking off the cuff on at least one night, three had incomplete questionnaire data, and four did not provide blood samples (one of whom also had a BMI value outside four standard deviations). Thus the analytic sample ranges from 231 to 234, specifically analyses involving questionnaire-reported conflictual life events and physical activity (N=231), inflammatory factors (N=231), HOMA-IR (N=232), nighttime BP (N=232), BMI percentile (N=234), daytime and 24-hour BP (N=234), and waist circumference (N=234).

Prior evidence in this sample (Jakubowski et al., 2014) indicated no relationship between napping (by either method) or nocturnal sleep and family socioeconomic status, as measured by total family score on the Hollingshead Four Factor Index (Hollingshead, 1975). Therefore family socioeconomic status was not included as a covariate in the present study. Age, sex, and race were used as covariates in all analyses. BMI percentile was used as a covariate in relevant analyses to adjust for overall obesity; this measure indicates the relative position of the adolescent's BMI among adolescents of the same sex and age using charts provided by the Centers for Disease Control (2011). Due to skewness, BMI percentile was square-root transformed after subtracting the BMI percentile value from 100 [square root (100 – BMI percentile)]. Analyses involving waist circumference were run within-gender, and did not include BMI percentile as a covariate, given the high correlation between these variables ( $r = .80$ ,  $p < .001$ ). Differences in sociodemographic variables, napping and nocturnal sleep (by actigraphy and diary measures), CV risk factors, health behaviors, and interpersonal conflict were examined by gender and race, as well as the interaction of gender and race, using 2 x 2 analysis of variance for continuous data and Chi square statistics for categorical data. Unadjusted

correlations were conducted between nocturnal sleep and napping variables (by actigraphy and diary measures) and CV risk factors. P-values were considered statistically significant at  $<.05$ .

#### **9.4.1 Primary aim**

To address whether napping was related to CV risk, linear regressions were conducted to measure associations between CV risk factors and four separate nap variables: actigraphy- and diary-measured proportion of days napped and actigraphy- and diary-measured average minutes napped across the study period. All analyses adjusted for age, sex, and race.

*Hypothesis 1: Napping would be associated with elevated CV risk.* To address Hypothesis 1, CV risk factors were separately regressed onto nap variables.

*Hypothesis 2: Napping would be associated with elevated CV risk, after adjustment for short nocturnal sleep.* CV risk factors were separately regressed onto the four nap variables with nocturnal sleep (actigraphy- or diary-measured average nocturnal sleep across the week-long sampling period, using the same method as the nap variable of interest) included as a covariate.

*Hypothesis 3: Napping would be associated with elevated CV risk, after adjustment for short nocturnal sleep and BMI percentile.* CV risk factors were separately regressed onto the four nap variables, with both average nocturnal sleep and BMI percentile included as covariates.

#### **9.4.2 Exploratory aims**

If results from the primary aim suggested a relationship between napping and elevated CV risk, subsequent exploratory analyses were conducted.

*Exploratory Hypothesis 1: The effect of napping on CV risk would be stronger in adolescents reporting decreased physical activity.* CV risk factors were regressed onto napping and physical activity variables, as well as the interaction between napping and physical activity. All analyses were adjusted for age, sex, and race. Subsequent analyses separately entered nocturnal sleep and BMI percentile in the model.

*Exploratory Hypothesis 2: The effect of napping on CV risk would be stronger in adolescents reporting increased sedentary behaviors.* CV risk factors were regressed onto napping and sedentary behavior variables, as well as the interaction between napping and sedentary behaviors. All analyses were adjusted for age, sex, and race. Subsequent analyses separately entered nocturnal sleep and BMI percentile in the model.

*Exploratory Hypothesis 3: The effect of napping on CV risk would be stronger in adolescents reporting increased interpersonal conflict.* CV risk factors were regressed onto napping and interpersonal conflict variables, as well as the interaction between napping and interpersonal conflict. All analyses were adjusted for age, sex, and race. Subsequent analyses separately entered nocturnal sleep and BMI percentile in the model.

### **9.4.3 Additional exploratory aims**

Given evidence that blacks and males in this sample obtained less nocturnal sleep, relative to whites and females (Matthews, Hall, & Dahl, 2014), and evidence that females in this sample napped more often and longer than males (Jakubowski et al., 2014), exploratory analyses included moderation by race and gender. Furthermore, considering the previously discussed evidence that there is a biological shift in sleep-wake behavior at the onset of puberty (e.g.,

Carskadon et al., 1980), we also explored whether the relationship between napping and CV risk was stronger in pre- versus post-pubertal males.

Mezick and colleagues (2012) found that obtaining more sleep at night was related to higher sleep:wake SBP ratio and lower nighttime BP, but not daytime BP, thus we aimed to explore the relationship between napping and separate BP values, particularly daytime and nighttime BP, as well as the sleep:wake SBP ratio.

Also in this sample, evidence suggests that adolescents obtain shorter sleep on weekdays versus weekends (Matthews, Hall, & Dahl, 2014), and those obtaining short sleep on weekdays demonstrate elevated CRP level (Hall, Lee, & Matthews, 2014). Consequently, the present study examined the relationship between napping and CV risk factors using average minutes napped on weekdays and weekends separately.

Finally, two sets of analyses were conducted using the sub-samples of adolescents who reported napping by actigraphy and those who reported napping by diary, to determine if the relationship between napping and CV risk factors differed when non-nappers were excluded.

## **10.0 RESULTS**

### **10.1 SAMPLE CHARACTERISTICS**

The analytic sample was composed of 62 black males, 68 black females, 48 white males, and 56 white females. Their average age was approximately 16 years (Table 1). The sample was from low to middle class as evidenced by their family Hollingshead scores (range: 10–54); black adolescents had higher family Hollingshead scores, relative to whites. The majority of the sample was advanced or post-pubertal (84%), although fewer males had gone through puberty (66%), relative to females (98%; Table 1).

**Table 1.** Characteristics of Overall Analytic Sample

	Total Sample	Black		White		Significant $F$ , $\chi^2$ values
		Male	Female	Male	Female	
N (%)	234	62 (26.5)	68 (29.1)	48 (20.5)	56 (23.9)	
Age, years ( $M$ , $SD$ )	15.7 (1.3)	15.7 (1.2)	15.6 (1.4)	15.7 (1.5)	15.6 (1.1)	
Family Hollingshead Total ( $M$ , $SD$ )	30.4 (11.5)	33.5 (11.8)	30.7 (11.5)	29.0 (11.6)	27.7 (10.3)	$R^{**}(1, 225)=6.3$
Pubertal Status (advanced or post-pubertal), N (%) <sup>a</sup>	193 (84%)	38 (61%)	66 (99%)	35 (73%)	55 (98%)	$\chi^2(1, N=231) = 41.3^{***}$
Smokers in last 30 days, N (%)	61 (26%)	13 (21%)	14 (21%)	14 (29%)	20 (36%)	$R^{**}(1, 227)=4.0$
<i>Actigraphy Measures</i>						
Nocturnal sleep duration, hours ( $M$ , $SD$ )	6.4 (0.8)	6.2 (0.8)	6.3 (0.6)	6.5 (0.7)	6.9 (0.9)	$G^{***}(1,230)=8.5$ $R^{***}(1,230)=23.3$
Proportion of days napped; ( $M$ , $SD$ )	0.36 (0.22)	0.31 (0.22)	0.43 (0.23)	0.31 (0.22)	0.35 (0.21)	$G^{***}(1,230)=8.2$
Average minutes napped across the study period (nappers only); $Mdn$ , (IQR)	20.7, (10.4, 36.4)	15.7, (8.0, 34.9)	29.0, (18.1, 40.5)	15.1, (7.5, 21.9)	22.4, (9.8, 36.4)	$G^{***}(1,207)=9.8$ $R^{**}(1,207)=5.5$
Average minutes napped across all weekdays (nappers only); $Mdn$ , (IQR)	17.4, (6.8, 34.6)	15.4, (7.4, 35.0)	28.2, (12.6, 40.1)	10.1, (3.1, 17.1)	23.2, (5.9, 39.3)	$G^{***}(1,207)=7.7$ $R^{*}(1,207)=3.5$
Average minutes napped across all weekend days (nappers only); $Mdn$ , (IQR)	16.0, (0.0, 40.0)	16.5, (0.0, 30.0)	24.0, (2.8, 59.3)	13.5, (0.0, 31.9)	8.5, (0.0, 37.3)	$G^{**}(1,207)=9.8$ $RxG^{*}(1,207)=2.9$
<i>Diary Measures</i>						
Nocturnal sleep duration, hours ( $M$ , $SD$ )	7.4 (0.9)	7.3 (0.9)	7.2 (0.9)	7.5 (0.83)	7.7 (1.0)	$R^{***}(1,230)=8.2$ $RxG^{*}(1,230)=3.5$
Proportion of days napped; $Mdn$ , (IQR)	0.28, (0.14, 0.43)	0.23, (0.14, 0.41)	0.29, (0.14, 0.41)	0.29, (0.14, 0.33)	0.29, (0.15, 0.43)	
Average minutes napped across the study period (nappers only); $Mdn$ , (IQR)	32.9, (17.4, 52.4)	30.2, (13.8, 53.5)	32.9, (19.4, 54.1)	30.0, (17.1, 51.3)	34.3, (23.6, 56.6)	

Average minutes napped across all weekdays (nappers only); <i>Mdn</i> , (IQR)	36.0, (13.0, 60.0)	31.0, (5.8, 64.9)	38.2, (11.8, 60.0)	29.6, (5.0, 45.0)	36.0, (22.5, 58.0)	
Average minutes napped across all weekend days (nappers only); <i>Mdn</i> , (IQR)	0.0, (0.0, 45.0)	0.0, (0.0, 40.0)	0.0, (0.0, 42.2)	0.0, (0.0, 45.0)	15.0, (0.0, 81.3)	
Proportion of days physically active ( <i>M</i> , <i>SD</i> )	0.42 (0.33)	0.50 (0.32)	0.33 (0.31)	0.49 (0.34)	0.36 (0.34)	$G^{***}(1,230)=12.5$
Proportion of days with interpersonal conflict, ( <i>M</i> , <i>SD</i> )	0.41 (0.30)	0.30 (0.29)	0.40 (0.27)	0.35 (0.28)	0.59 (0.30)	$G^{***}(1,230)=21.8$ $R^{***}(1,230)=10.4$ $RxG^*(1,230)=3.7$
<i>CV Risk Factors</i>						
BMI percentile ( <i>M</i> , <i>SD</i> )	78.8 (22.6)	78.9 (20.1)	82.7 (17.8)	80.7 (24.8)	72.2 (27.2)	$RxG^{**}(1,230)=4.3$
WC, <i>Mdn</i> , (IQR)	30.0, (27.0, 34.0)	30.0, (28.0, 32.0)	30.0, (27.0, 34.0)	33.0, (30.0, 38.8)	29.0, (26.0, 32.0)	$G^{***}(1,230)=10.9$ $RxG^{***}(1,230)=10.4$
24-hour systolic blood pressure ( <i>M</i> , <i>SD</i> )	118.9 (8.9)	121.9 (8.3)	117.6 (8.9)	119.6 (10.4)	116.4 (7.4)	$G^*(1,230)=10.7$
HOMA-IR; <i>Mdn</i> , (IQR)	3.7 (2.7, 4.8)	3.2 (2.4, 4.4)	4.1 (2.9, 5.2)	3.8 (2.7, 5.3)	3.6 (2.9, 4.6)	$RxG^*(1,228)=3.2$
hs-CRP; <i>Mdn</i> , (IQR)	0.6 (0.3, 1.6)	0.4 (0.2, 1.0)	0.7 (0.2, 2.4)	0.9 (0.4, 1.6)	0.7 (0.3, 1.6)	
IL-6; <i>Mdn</i> , (IQR)	1.0 (0.7, 1.9)	0.8 (0.5, 1.9)	1.0 (0.8, 1.8)	1.1 (0.7, 2.2)	1.2 (0.7, 1.8)	$R^{**}(1,227)=4.0$
<i>Questionnaire Measures</i>						
YRBSS Days physically active ( <i>M</i> , <i>SD</i> ) <sup>b</sup>	3.5 (2.3)	4.3 (2.4)	3.2 (2.3)	4.0 (1.9)	2.5 (2.1)	$G^{***}(1,227)=19.5$ $R^*(1,227)=2.9$
YRBSS Sedentary Behaviors ( <i>M</i> , <i>SD</i> ) <sup>c</sup>	2.9 (1.5)	3.2 (1.3)	2.8 (1.5)	3.1 (1.5)	2.5 (1.4)	$G^{**}(1,227)=6.2$
LEQ-A No. of Conflictual Life Events ( <i>M</i> , <i>SD</i> )	2.2 (1.5)	1.7 (1.3)	2.2 (1.4)	2.3 (1.5)	2.9 (1.5)	$G^{***}(1,227)=8.9$ $R^{***}(1,227)=10.3$
CES-D	15.4 (8.6)	11.6 (6.5)	15.6 (8.1)	14.9 (8.8)	19.7 (9.1)	$G^{***}(1,227)=16.3$ $R^{***}(1,227)=11.7$

*Note.* IQR = Interquartile Range (25<sup>th</sup>, 75<sup>th</sup> percentiles); BMI = body mass index; WC = sex-standardized waist circumference; SBP = systolic blood pressure; DBP = diastolic blood pressure; YRBSS = Centers for Disease Control and Prevention Youth Risk Behavior Surveillance System; LEQ-A = The Life Events Questionnaire-Adolescent; CES-D = Center for Epidemiological Studies–Depression.

<sup>a</sup> Pubertal stage measured using the Peterson Development Scale (PDS). <sup>b</sup>Days physically active  $\geq 60$  min in last week. <sup>c</sup>YRBSS Sedentary Behavior represents a 6-point scale of the average number of hours engaged in sedentary behaviors on a typical school day; *M* (*SD*) reported in table do not directly correspond to hours; For example, “2.9” = approximately 1 hour per day and “3” = approximately 2 hours per day. \* $p < .1$ . \*\* $p < .05$ . \*\*\* $p < .01$ .

## **10.2 NOCTURNAL SLEEP AND NAPPING CHARACTERISTICS**

On average, adolescents slept 6.4 (range: 4.3–9.2) hours at night by actigraphy, and 7.4 hours (range: 4.8–9.6) by daily diary report. As reported elsewhere based on the full sample (Matthews, Hall, & Dahl, 2014), there were significant race and gender differences in actigraphy-measured nocturnal sleep duration, with black adolescents and males demonstrating shorter duration, relative to white adolescents and females. Significant race differences emerged for diary sleep duration, with blacks reporting shorter nocturnal sleep duration (Table 1).

Napping was a common behavior (Table 1; left column). Ninety percent had at least one actigraphy-assessed nap and 61% reported at least one diary-assessed nap. Adolescents napped on average 36% (range: 0–100%) of the days by actigraphy and 18% (range: 0–86%) of the days by diary. On average, adolescents napped 24 minutes per day by actigraphy across the week-long study period, and 26 minutes per day by diary. There were significant race and gender differences with regard to actigraphy-assessed napping. Females napped more days by actigraphy, relative to males. Females and black adolescents demonstrated significantly increased average actigraphy-assessed minutes napped across the study period, relative to males and white adolescents. Diary-reported napping (proportion of days napped or average minutes napped across the study period) did not vary by race or gender.

Overall, the length of diary-reported naps on weekdays were significantly longer than those reported on weekends, while there were no differences in the length of actigraphy-assessed naps on weekdays versus weekends (analysis not shown). Females also demonstrated more napping on both weekdays and weekends (analysis not shown).



### **10.3 CV RISK FACTORS**

Overall sample values for CV risk factors can be found in Table 1; left column. On average, adolescents were overweight and in the normal range for 24-hour average SBP. They demonstrated high values for HOMA-IR and hs-CRP. Approximately one-quarter of the sample reported smoking cigarettes in the past thirty days.

As reported elsewhere based on the full sample (Matthews, Hall, & Dahl, 2012), results indicated that black females demonstrated the highest BMI percentile, relative to all other groups. There was a significant effect of gender and also a significant race by gender interaction for waist circumference. Males demonstrated larger waist circumference relative to females, particularly white males, compared to all other groups. Finally, significant race differences emerged for IL-6, such that white adolescents demonstrated higher IL-6 values, relative to black adolescents (Table 1). More white adolescents reported smoking (33%), relative to blacks (21%).

### **10.4 HEALTH BEHAVIORS**

#### **10.4.1 Physical activity**

Adolescents reported being physically active on 42% (range: 0–100%) of study days in their diaries; males reported more days on which they were physically active (50%), relative to females (35%; Table 1). With regard to questionnaire-report of physical activity, on average, adolescents reported being physically active for at least 60 minutes per day on 3.5 days during the past week (range: 0–7 days). As reported elsewhere based on the full sample (Matthews,

Hall, & Dahl, 2014), significant gender differences emerged in number of days engaged in physical activity, such that males reported being physically active on more days than females (4.2 versus 2.9, respectively).

#### **10.4.2 Sedentary behaviors**

Adolescents engaged in 1-2 hours of sedentary behaviors on an average school day (range: 0–5 hours per day). On average, males spent more hours in sedentary activities, relative to females; see Table 1.

#### **10.4.3 Interpersonal conflict**

Adolescents reported interpersonal conflict in their daily diaries on 41% of sampling days (range: 0–100%). Significant race and gender differences emerged for interpersonal conflict, such that whites and females reported interpersonal conflict in their diaries on more days, relative to blacks and males (Table 1).

With regard to questionnaire report of conflictual life events, adolescents reported an average of 2.2 events in the past 12 months (range: 0–5). As reported elsewhere based on the larger sample (Low, Matthews, & Hall, 2013), whites and females reported more interpersonal conflict life events, relative to blacks and males (Table 1).

#### **10.4.4 Depression**

The overall CES-D score for the sample was 15.4. Self-report of depressive symptoms was significantly higher in whites and females, relative to blacks and males (Table 1).

### **10.5 BIVARIATE CORRELATIONS**

Correlations among all variables included in primary and exploratory aims are presented in Table 2. The longer the average nocturnal sleep duration measured by actigraphy, the fewer days napped by both actigraphy and diary, and the shorter the naps by both methods. The proportion of days napped and length of naps on napping days were highly correlated by both actigraphy and diary report. Naps estimated by actigraphy and diary were highly correlated. More days napped by actigraphy was associated with longer average actigraphy-assessed naps, shorter diary-reported average nocturnal sleep, more days napped and longer average naps by diary, and more days with diary-reported interpersonal conflict. Average minutes napped across the study period by actigraphy was associated with shorter diary-reported nocturnal sleep duration, more days napped and longer average naps by diary, more days with diary-reported interpersonal conflict, and greater HOMA-IR values.

Longer average nocturnal sleep duration measured by actigraphy was related to fewer days engaged in at least 60 minutes of physical activity in the past week, fewer average hours of sedentary behaviors, and lower 24-hour SBP. Longer diary-reported nocturnal sleep duration was associated with fewer days napped and shorter average naps by diary report, fewer days engaged in physical activity for 60 minutes or longer, and smaller HOMA-IR values. Diary-reported

proportion of days napped was associated with longer average naps by diary report and more days with diary-reported interpersonal conflict.

Diary-reported interpersonal conflict was associated with more conflictual life events, and lower 24-hour SBP. Higher depression was associated with both conflictual life events and diary-reported interpersonal conflict. More days engaged in physical activity for at least 60 minutes in the past week was related to fewer conflictual life events, as well as lower HOMA-IR and higher 24-hour SBP values. Greater waist circumference and BMI percentile were both associated with greater HOMA-IR, hs-CRP, and IL-6 values. Waist circumference and BMI percentile were highly correlated, as were hs-CRP and IL-6 values.

**Table 2.** Correlations Among Primary Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
<u>Actigraphy</u>																	
1 Nocturnal duration	--																
2 Proportion of days napped	.23**	--															
3 Average minutes napped	.26**	.68**	--														
<u>Diary</u>																	
4 Nocturnal duration	.59**	.23**	.27**	--													
5 Proportion of days napped	.16*	.44*	.42**	.19**	--												
6 Average minutes napped	.17*	.43**	.52**	.16*	.81**	--											
7 Physical activity	.02	.10	.09	.06	.08	.07	--										
8 Proportion of days with inter-personal conflict	.04	.15*	.16*	.07	.16*	.13	.05	--									
<u>Questionnaire</u>																	
9 YRBSS physical activity <sup>a</sup>	.17**	.05	.01	.18**	.01	.02	.05	.04	--								
10 YRBSS Sedentary behaviors <sup>b</sup>	.14*	.02	.03	.12	.02	.03	.03	.02	.06	--							
11 LEQ-A	.04	.09	.05	.05	.03	.05	.03	.23**	.14*	.12	--						
12 CES-D	.01	.07	.09	.09	.05	.05	.13	.37**	.15*	.05	.36**	--					

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
<u>CV Risk Factors</u>																	
13 WC	.00	.07	.05	.05	.12	.13	.00	.06	.05	.08	.01	.02	--				
14 HOMA-IR	.13	.12	.20**	.17**	.10	.05	.08	.05	.14*	.03	.05	.08	.56**	--			
15 hs-CRP	.02	.05	.05	.01	.03	.03	.06	.07	.08	.10	.07	.13	.25**	.11	---		
16 IL-6	.07	.09	.06	.07	.01	.02	.03	.04	.03	.00	.09	.00	.25**	.07	.48*	--	
17 24-hour SBP	.22**	.11	.09	.05	.01	.06	.05	.16*	.16*	.03	.07	.11	.02	.06	.12	.01	--
18 BMI %	.05	.08	.05	.05	.08	.10	.05	.01	.09	.07	.03	.06	.80**	.39**	.16*	.18**	.01

*Note.* Proportion of days napped = Proportion of days napped across seven-day study period; Average minutes napped = Average minutes napped across seven-day study period; YRBSS = Centers for Disease Control and Prevention Youth Risk Behavior Surveillance System; LEQ-A = The Life Events Questionnaire – Adolescent; CES-D = WC = sex-standardized waist circumference; HOMA-IR = homeostatic model assessment of insulin resistance; hs-CRP = high sensitivity C-reactive protein; IL-6 = interleukin 6; SBP = systolic blood pressure; BMI % = Body Mass Index Percentile

<sup>a</sup>Days physically active  $\geq 60$  min in last week. <sup>b</sup>Average number of hours engaged in sedentary behaviors on a typical school day.

\*  $p < .05$ . \*\*  $p < .01$ .

## 10.6 PRIMARY AIM

The primary aim of the present study was to test whether napping was associated with elevated CV risk factors. The results for the three hypotheses are in Table 3. It was hypothesized that napping would be associated with elevated CV risk, after adjustment for age, race, and gender (*Hypothesis 1*). This hypothesis was not supported for any CV risk factor by any indicator of napping. The second hypothesis, that napping would be associated with elevated CV risk, independent of short nocturnal sleep (*Hypothesis 2*) was also not confirmed. Contrary to hypothesis, more days napped by actigraphy was associated with *lower* 24-hour SBP, after adjustment for average actigraphy-assessed nocturnal sleep. There was a trend for increased actigraphy-assessed proportion of days napped to be related to higher IL-6, after adjustment for average actigraphy-assessed nocturnal sleep. There were no associations between actigraphy- or diary-measured napping variables and waist circumference, HOMA-IR, or hs-CRP for Hypothesis 2. The third hypothesis, that napping would be associated with elevated CV risk after adjustment for short nocturnal sleep and BMI (*Hypothesis 3*), was confirmed for IL-6, but not for 24-hour average SBP. Increased actigraphy-assessed proportion of days napped was associated with higher IL-6 (see Table 3); results from the full multivariate model indicated that white adolescents ( $b = -.20$ ,  $SE = .09$ ,  $p = .034$ ) and those with a higher BMI percentile ( $b = -.11$ ,  $SE = .02$ ,  $p < .001$ ) demonstrated increased IL-6 values. Contrary to hypothesis, more days napped by actigraphy was associated with *lower* 24-hour SBP (Table 3); results from the full multivariate model indicated that females ( $b = -2.68$ ,  $SE = 1.16$ ,  $p = .022$ ) and adolescents who obtained more

nocturnal sleep ( $b = -2.27$ ,  $SE = .77$ ,  $p = .004$ ) demonstrated decreased 24-hour SBP. There were no associations between actigraphy- or diary-measured napping and waist circumference, HOMA-IR, or hs-CRP for Hypothesis 3.



**Table 3.** Regression Coefficients for Full Multivariate Model Testing Relationships Between Napping and CV Risk Factors

	<b>Sex-standardized WC</b>	<b>HOMA-IR</b>	<b>hs-CRP</b>	<b>IL-6</b>	<b>24-hr average SBP</b>
<i>Actigraphy-assessed napping</i>					
<b>Proportion of days napped</b>	-.12 (.30)	.21 (.13)	-.04 (.39)	.31 (.21)	-4.34 (2.60)
Hypothesis 1 <sup>a</sup>	-.11 (.31)	.18 (.13)	-.02 (.40)	.33 (.21)	-3.21 (2.58)
Hypothesis 2 <sup>b</sup>	-.10 (.32)	.09 (.14)	-.03 (.42)	.38 (.22)*	-5.19 (2.63)**
Hypothesis 3 <sup>c</sup>	N/A	-.10 (.01)	-.27 (.03)	.49 (.21)**	-5.29 (2.64)**
<b>Average minutes napped across the week</b>	-.00 (.06)	.04 (.02)	-.02 (.07)	.04 (.04)	-.59 (.48)
Hypothesis 1 <sup>a</sup>	.00 (.06)	.03 (.03)	.00 (.08)	.04 (.04)	-.26 (.49)
Hypothesis 2 <sup>b</sup>	.00 (.06)	.01 (.03)	.00 (.08)	.05 (.04)	-.60 (.49)
Hypothesis 3 <sup>c</sup>	N/A	.03 (.02)	.03 (.07)	.06 (.04)	-.61 (.50)
<i>Diary-assessed napping</i>					
<b>Proportion of days napped</b>	-.26 (.24)	.10 (.11)	-.20 (.32)	-.07 (.17)	-.43 (2.10)
Hypothesis 1 <sup>a</sup>	-.28 (.25)	.07 (.11)	-.31 (.32)	-.11 (.18)	1.15 (2.11)
Hypothesis 2 <sup>b</sup>	-.34 (.26)	.00 (.11)	-.39 (.34)	-.10 (.18)	.98 (2.17)
Hypothesis 3 <sup>c</sup>	N/A	.06 (.10)	-.21 (.30)	-.04 (.17)	.94 (2.18)
<b>Average minutes napped across the week</b>	-.05 (.04)	.01 (.02)	-.04 (.05)	-.01 (.03)	-.14 (.33)
Hypothesis 1 <sup>a</sup>	-.05 (.04)	.01 (.02)	-.06 (.05)	-.02 (.03)	.08 (.33)
Hypothesis 2 <sup>b</sup>	-.06 (.04)	-.00 (.02)	-.07 (.05)	-.01 (.03)	.04 (.34)
Hypothesis 3 <sup>c</sup>	N/A	.01 (.02)	-.04 (.05)	-.003 (.03)	.04 (.34)

*Note.* Values reflect unstandardized estimate (standard error); WC = waist circumference; HOMA-IR = homeostatic model assessment of insulin resistance; hs-CRP = high sensitivity C-reactive protein; IL-6 = interleukin 6; SBP = systolic blood pressure.

<sup>a</sup>Adjustment for age, race, and gender. <sup>b</sup>Adjustment for age, race, gender, and average nocturnal sleep (measured by the same method as napping variable of interest). <sup>c</sup>Adjustment for age, race, gender, average nocturnal sleep (measured by the same method as napping variable of interest), and BMI percentile.

\*  $p < .1$ . \*\* $p < .05$ .

## 10.7 EXPLORATORY AIMS

Additional exploratory analyses were conducted to investigate potential moderators of the relationship between napping and CV risk. *Exploratory Aim 1* aimed to test whether the effect of napping on CV risk was moderated by physical activity. It was hypothesized that the effect of napping on CV risk would be strongest in adolescents who reported decreased physical activity. The relationship between proportion of days napped by actigraphy and sex-standardized waist circumference varied as a function of questionnaire-reported physical activity ( $b = -.27$ ,  $SE = .14$ ,  $p = .046$ ; Table 4) after adjustment for nocturnal sleep, although no simple slopes were significantly different from zero (Figure 2). Additionally, the relationship between average minutes napped by diary and 24-hour SBP varied as a function of proportion of days with diary-reported physical activity ( $b = 2.12$ ,  $SE = .97$ ,  $p = .030$ ; Table 4) after adjustment for nocturnal sleep and BMI percentile, however no simple slopes were significantly different from zero (Figure 3). The relationship between napping and waist circumference did not vary by diary-reported physical activity, while the relationship between napping (by either method) and 24-hour SBP did not vary by questionnaire-reported physical activity (Table 4). Also against expectation, physical activity (measured by diary or questionnaire) did not moderate the association between napping (by actigraphy or diary) and HOMA-IR, hs-CRP, or IL-6 (Table 4).

**Table 4.** Interaction Terms Between Physical Activity Variables and Napping in Relationship to CV Risk Factors

	<b>Sex-standardized WC<sup>a</sup></b>	<b>HOMA-IR<sup>b</sup></b>	<b>hs-CRP<sup>b</sup></b>	<b>IL-6<sup>b</sup></b>	<b>24-hr average SBP<sup>b</sup></b>
<i>Questionnaire physical activity (YRBSS) * napping</i>					
<b>Actigraphy-assessed proportion of days napped</b>	-.27 (.14)**	-.05 (.05)	-.06 (.16)	.16 (.09)*	-.13 (1.14)
<b>Actigraphy-assessed average minutes napped across the week</b>	-.01 (.03)	.00 (.01)	-.01 (.03)	.02 (.02)	.02 (.21)
<b>Diary-assessed proportion of days napped</b>	-.05 (.11)	.02 (.04)	.03 (.13)	.06 (.07)	.18 (.93)
<b>Diary-assessed average minutes napped across the week</b>	-.01 (.02)	.00 (.01)	.02 (.02)	.01 (.01)	.06 (.14)
<i>Diary-reported physical activity * napping</i>					
<b>Actigraphy-assessed proportion of days napped</b>	-.90 (.94)	-.37 (.34)	-.76 (1.09)	-.02 (.61)	5.02 (7.80)
<b>Actigraphy-assessed average minutes napped across the week</b>	.03 (.18)	-.08 (.07)	-.22 (.21)	-.00 (.12)	2.53 (1.48)*
<b>Diary-assessed proportion of days napped</b>	-.04 (.75)	-.17 (.28)	-.84 (.87)	-.05 (.50)	9.98 (6.32)
<b>Diary-assessed average minutes napped across the week</b>	-.02 (.12)	-.01 (.04)	-.10 (.14)	.03 (.08)	2.12 (.97)**

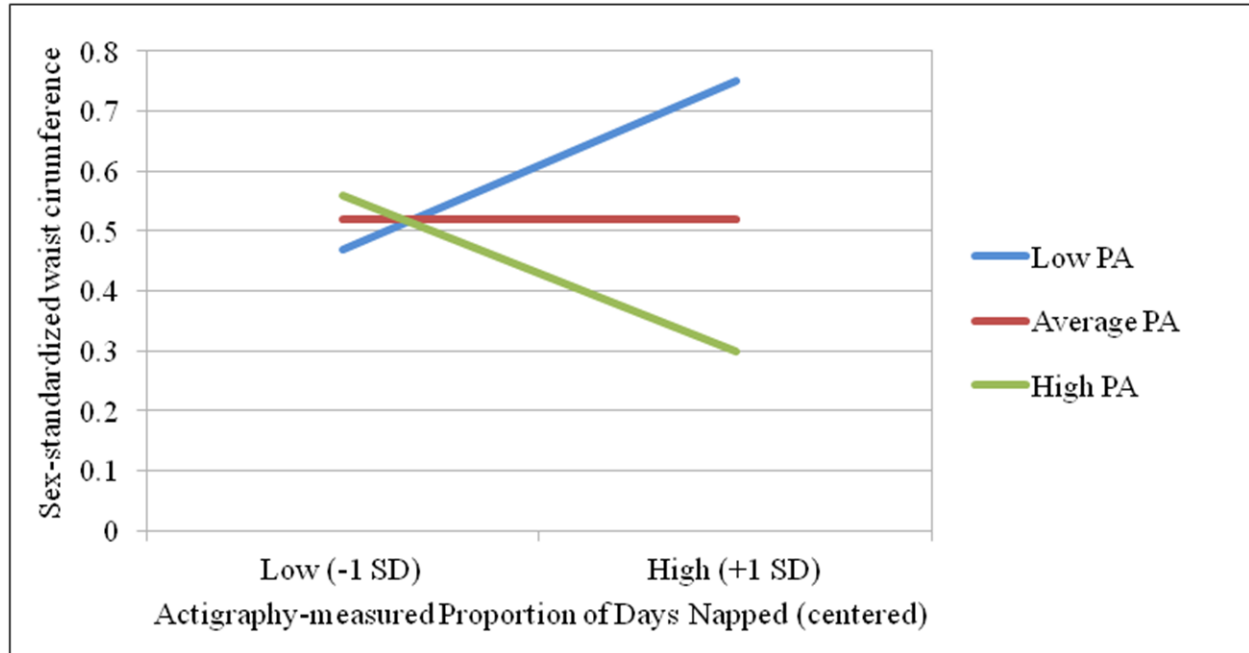
*Note.* Values reflect unstandardized estimate (standard error) for the interaction term of napping X physical activity. Questionnaire-reported physical activity, daily diary-reported physical activity, and all actigraphy- and diary-assessed napping variables have been mean-centered. YRBSS = Centers for Disease Control and Prevention Youth Risk Behavior Surveillance System; WC = waist circumference; HOMA-IR = homeostatic model assessment of insulin resistance; hs-CRP = high sensitivity C-reactive protein; IL-6 = interleukin 6; SBP = systolic blood pressure.

<sup>a</sup>Adjustment for age, race, gender, and average nocturnal sleep (measured by the same method as napping variable of interest).

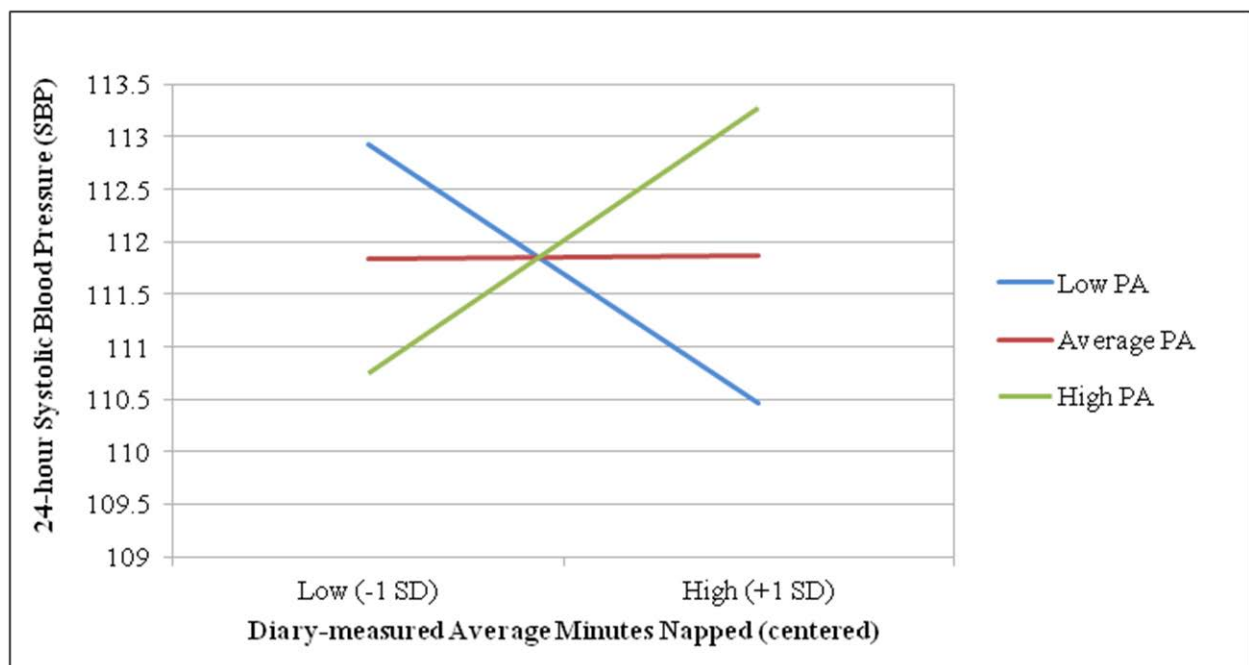
<sup>b</sup>Adjustment for age, race, gender, average nocturnal sleep (measured by the same method as napping variable of interest), and BMI percentile.

\*  $p < .1$ . \*\* $p < .05$ .

**Figure 2.** Simple Slopes of Sex-standardized Waist Circumference on Actigraphy-measured Proportion of Days Napped at Low, Average, and High Levels of Questionnaire-reported Physical Activity (PA)



**Figure 3.** Simple Slopes of 24-hour Systolic Blood Pressure on Diary-measured Average Minutes Napped at Low, Average, and High Levels of Diary-reported Physical Activity (PA)



*Exploratory Aim 2* aimed to test whether the effect of napping on CV risk was moderated by sedentary behaviors. Contrary to prediction, increased sedentary behaviors did not moderate the relationship between napping (by actigraphy or diary measures) and waist circumference, HOMA-IR, hs-CRP, IL-6, or 24-hour SBP values (Table 5).

**Table 5.** Interaction Terms Between Sedentary Behaviors and Napping in Relationship to CV Risk Factors

	<b>Sex-standardized WC<sup>a</sup></b>	<b>HOMA-IR<sup>b</sup></b>	<b>hs-CRP<sup>b</sup></b>	<b>IL-6<sup>b</sup></b>	<b>24-hr average SBP<sup>b</sup></b>
<i>Sedentary behaviors (YRBSS) * napping</i>					
<b>Actigraphy-assessed proportion of days napped</b>	-.21 (.22)	-.05 (.08)	.01 (.25)	-.21 (.14)	-3.33 (1.79)*
<b>Actigraphy-assessed average minutes napped across the week</b>	-.04 (.04)	-.01 (.01)	-.04 (.04)	-.03 (.03)	-.47 (.32)
<b>Diary-assessed proportion of days napped</b>	-.01 (.17)	-.03 (.06)	.08 (.19)	-.12 (.11)	-.88 (1.45)
<b>Diary-assessed average minutes napped across the week</b>	-.02 (.03)	-.01 (.01)	-.02 (.03)	-.02 (.02)	-.22 (.23)

*Note.* Values reflect unstandardized estimate (standard error) for the interaction term of napping X sedentary behaviors. Sedentary behaviors and all actigraphy- and diary-assessed napping variables have been mean-centered. YRBSS = Centers for Disease Control and Prevention Youth Risk Behavior Surveillance System; WC = waist circumference; HOMA-IR = homeostatic model assessment of insulin resistance; hs-CRP = high sensitivity C-reactive protein; IL-6 = interleukin 6; SBP = systolic blood pressure.

<sup>a</sup>Adjustment for age, race, gender, and average nocturnal sleep (measured by the same method as napping variable of interest).

<sup>b</sup>Adjustment for age, race, gender, average nocturnal sleep (measured by the same method as napping variable of interest), and BMI percentile.

\*  $p < .1$ . \*\* $p < .05$ .

**Exploratory Aim 3** aimed to test whether the effect of napping on CV risk was strongest in adolescents who reported increased interpersonal conflict. Contrary to prediction, increased actigraphy-assessed average minutes napping was *protective* for 24-hour SBP values among those with high levels of life events conflict (Table 6). The relationship between average minutes napped by actigraphy and 24-hour average SBP varied as a function of life events conflict ( $b = -.76$ ,  $SE = .31$ ,  $p = .015$ ), after adjustment for nocturnal sleep and BMI percentile. At high levels of conflict (+1 SD), napping was associated with lower levels of 24-hr SBP, while at low (-1SD) or average levels of conflict, there was no effect of napping on 24-hr SBP (Figure 4). Results held after further adjustment for depressive symptoms ( $b = -.79$ ,  $SE = .31$ ,  $p = .012$ ). Also against expectation, increased diary-reported average minutes napping was *protective* for 24-hour SBP values ( $b = -.47$ ,  $SE = 0.22$ ,  $p = .036$ ; Table 6) after adjustment for nocturnal sleep and BMI percentile, although no simple slopes were significantly different from zero (Figure 5). Results held after adjustment for depressive symptoms ( $b = -.49$ ,  $SE = .22$ ,  $p = .028$ ). The relationship between actigraphy-assessed or diary-reported napping and 24-hour SBP did not vary by interpersonal conflict reported in the daily diary (Table 6). Additionally, conflict (by diary or questionnaire) did not emerge as a moderator of the association between napping (by actigraphy or diary) and waist circumference, HOMA-IR, hs-CRP, or IL-6 values (Table 6).

**Table 6.** Interaction Terms Between Interpersonal Conflict Variables and Napping in Relationship to CV Risk Factors

	<b>Sex-standardized WC<sup>a</sup></b>	<b>HOMA-IR<sup>b</sup></b>	<b>hs-CRP<sup>b</sup></b>	<b>IL-6<sup>b</sup></b>	<b>24-hr average SBP<sup>b</sup></b>
<i>Life events conflict (LEQ-A) * napping</i>					
<b>Actigraphy-assessed proportion of days napped</b>	-.31 (.21)	-.11 (.08)	-.24 (.24)	-.16 (.14)	-2.89 (1.73)*
<b>Actigraphy-assessed average minutes napped across the week</b>	-.04 (.04)	-.02 (.01)	-.05 (.04)	-.02 (.03)	-.76 (.31)**
<b>Diary-assessed proportion of days napped</b>	-.02 (.07)	-.05 (.06)	-.22 (.19)	-.21 (.11)*	-2.68 (1.41)*
<b>Diary-assessed average minutes napped across the week</b>	.01 (.03)	-.01 (.01)	-.02 (.03)	-.03 (.02)*	-.47(.22)**
<i>Diary-reported interpersonal conflict * napping</i>					
<b>Actigraphy-assessed proportion of days napped</b>	.49 (.97)	-.39 (.35)	-.54 (1.11)	-.53 (.62)	1.55 (8.00)
<b>Actigraphy-assessed average minutes napped across the week</b>	-.02 (.19)	-.02 (.07)	.05 (.22)	.00 (.12)	-1.44 (1.58)
<b>Diary-assessed proportion of days napped</b>	.65 (.83)	.15 (.30)	.31 (.96)	.14 (.54)	-2.78 (7.00)
<b>Diary-assessed average minutes napped across the week</b>	.13 (.13)	.02 (.05)	.13 (.15)	.04 (.09)	-1.12 (1.12)

*Note.* Values reflect unstandardized estimate (standard error) for the interaction term of napping X interpersonal conflict. Life events conflict, diary reported interpersonal conflict, and all actigraphy- and diary-assessed napping variables have been mean-centered. LEQ-A = The Life Events Questionnaire – Adolescent; WC = waist circumference; HOMA-IR = homeostatic model assessment of insulin resistance; hs-CRP = high sensitivity C-reactive protein; IL-6 = interleukin 6; SBP = systolic blood pressure.

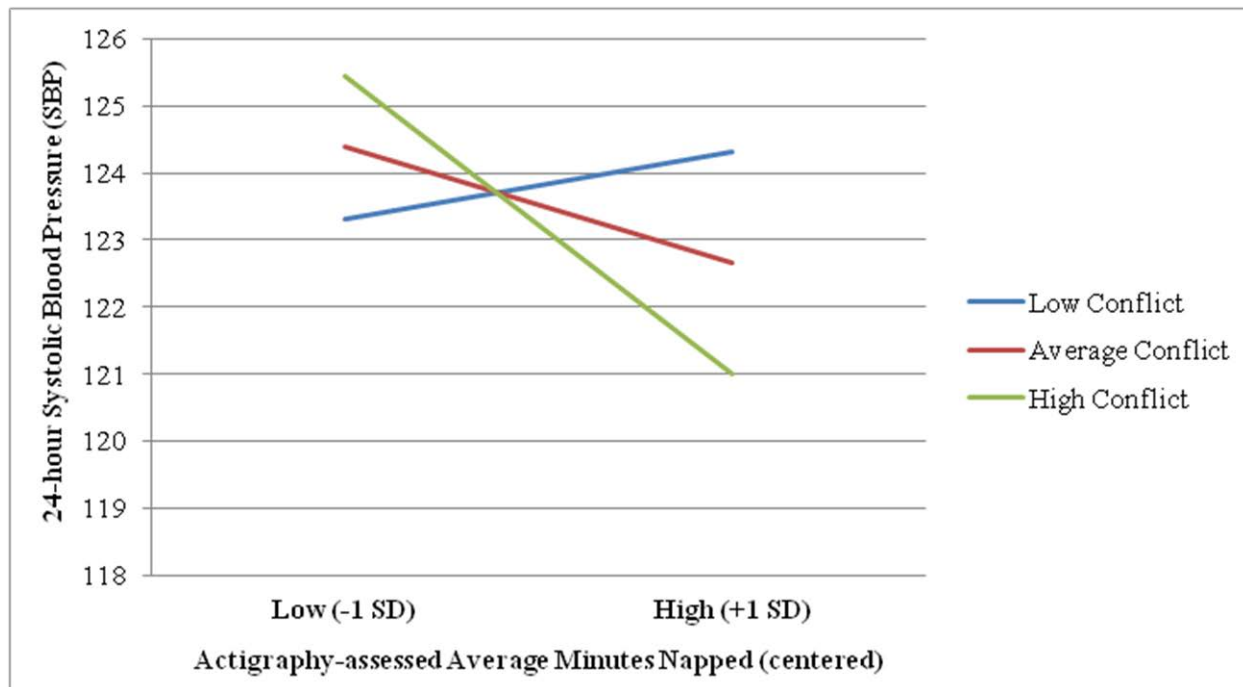
<sup>a</sup>Adjustment for age, race, gender, and average nocturnal sleep (measured by the same method as napping variable of interest).

<sup>b</sup>Adjustment for age, race, gender, average nocturnal sleep (measured by the same method as napping variable of interest), and BMI percentile.

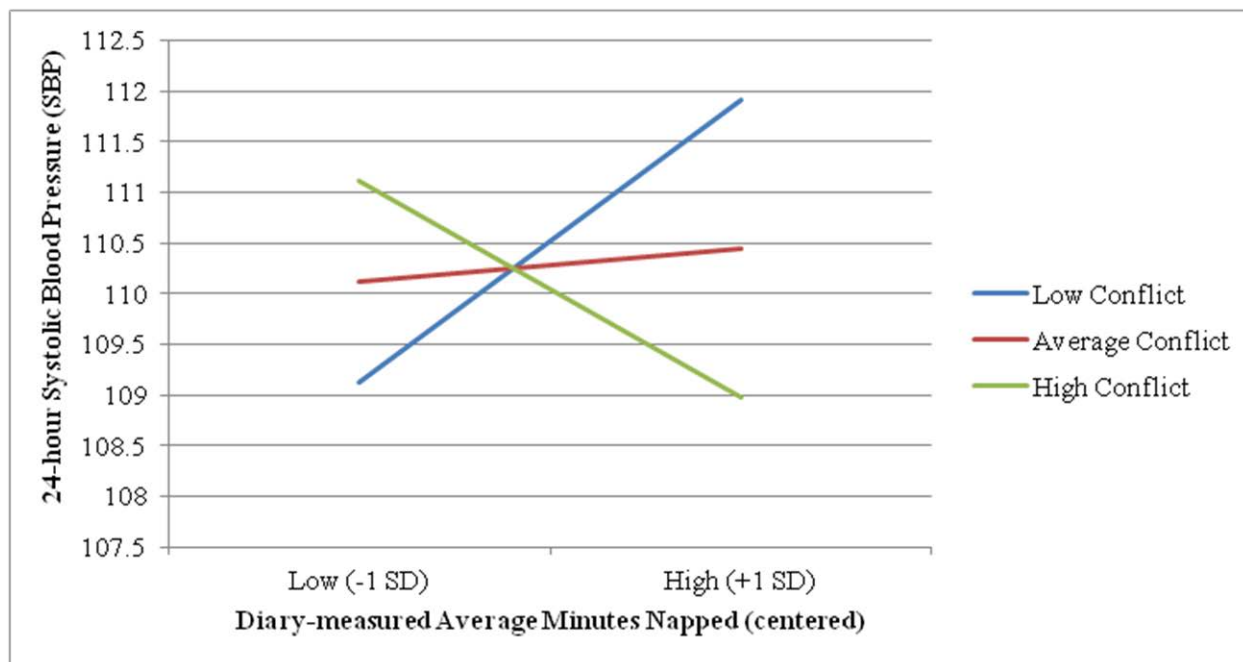
\*  $p < .1$ . \*\* $p < .05$ .



**Figure 4.** Simple Slopes of 24-hour Systolic Blood Pressure on Actigraphy-assessed Average Minutes Napped at Low, Average, and High Levels of Conflictual Life Events



**Figure 5.** Simple Slopes of 24-hour Average Systolic Blood Pressure on Diary-measured Average Minutes Napped at Low, Average, and High Levels of Conflictual Life Events



## **10.8 OTHER RELEVANT ANALYSES**

### **10.8.1 Moderation by gender and race**

We tested whether the relationship between napping and CV risk varied by race or gender. For the 40 tests, only one was significant. Results indicated an interaction between gender and actigraphy-assessed proportion of days napped on age- and sex-standardized waist circumference ( $b=1.21$ ,  $SE = 0.62$ ,  $p = .051$ ), after adjustment for nocturnal sleep. Sex-stratified analyses revealed that more days napped by actigraphy was associated with increased waist circumference in males only ( $b = -.79$ ,  $SE = .45$ ,  $p = .079$ ).

### **10.8.2 Moderation by pubertal status**

In males only, we tested the interaction of pubertal status and napping on CV risk. Out of the 20 tests, results indicated one trend for increased average minutes napped by diary to predict IL-6 ( $b = -.16$ ,  $SE = .09$ ,  $p = .070$ ). More diary napping was associated with greater IL-6 in males in the beginning or middle stages of puberty; in contrast, in advanced or post-pubertal males, more napping was associated with lower IL-6.

### **10.8.3 Additional blood pressure variables**

Table 7 displays relationships between napping and additional blood pressure variables, after adjustment for average nocturnal sleep and BMI percentile. Similar to the results for SBP, more days napped by actigraphy predicted lower 24-hr average DBP and daytime DBP; results from the full multivariate models suggested that females demonstrated higher 24-hr average DBP and daytime DBP values, while heavier adolescents and those obtaining more sleep at night demonstrated lower values. More days napped by actigraphy also predicted lower daytime SBP; females, heavier adolescents, and those obtaining more sleep at night also demonstrated lower values. More days napped by actigraphy also predicted increased SBP ratio (Table 7); females also demonstrated higher values.

More average minutes napped by actigraphy predicted lower 24-hour average DBP and daytime DBP (Table 7). In results from the full multivariate models, females demonstrated higher 24-hr average DBP and daytime DBP, while heavier adolescents and adolescents who obtained more nocturnal sleep duration demonstrated lower DBP values. More diary-reported minutes napped was associated with higher SBP ratio; females and black adolescents also demonstrated higher values. Napping was unrelated to average nighttime SBP or DBP (Table 7).

**Table 7.** Full Multivariate Model Relationship Between Napping and Additional Blood Pressure Variables

	<b>24-hr average DBP</b>	<b>Daytime SBP</b>	<b>Daytime DBP</b>	<b>Nighttime SBP</b>	<b>Nighttime DBP</b>	<b>SBP Night:Day Ratio</b>
<i>Actigraphy-assessed napping</i>						
<b>Proportion of days napped</b>	-3.90 (1.69)**	-6.75 (2.75)**	-4.84 (1.81)**	-2.06 (2.73)	-.90 (1.66)	.04 (.02)**
<b>Average minutes napped across the week</b>	-.82 (.31)**	-.71 (.52)	-.91 (.34)**	-2.06 (2.73)	-.33 (.31)	.00 (.00)
<i>Diary-assessed napping</i>						
<b>Proportion of days napped</b>	-.20 (1.40)	.36 (2.25)	-.76 (1.49)	3.03 (2.25)	1.96 (1.35)	.03 (.01)*
<b>Average minutes napped across the week</b>	-.18 (.22)	-.07 (.36)	-.26 (.23)	.49 (.35)	.25 (.21)	.01 (.00)**

*Note.* Values reflect unstandardized estimate (standard error). All analyses adjusted for age, sex, race, average nocturnal sleep (measured by the same method as napping variable of interest), and BMI percentile. SBP = systolic blood pressure; DBP = diastolic blood pressure.

\*  $p < .1$ . \*\* $p < .05$ .

#### **10.8.4 Weekdays versus weekends**

We tested if there was variability in findings by average minutes napped on weekdays or weekends. For the 40 tests conducted using the full multivariate model, only one was significant. As actigraphy-assessed napping on weekdays increased, IL-6 increased ( $b = .07$ ,  $SE = .03$ ,  $p = .037$ ), adjusting for average weekday nocturnal sleep and BMI percentile. Neither diary-reported weekday minutes napped, nor weekend minutes napped (by actigraphy or diary), predicted CV risk factors.

#### **10.8.5 Analyses excluding non-nappers**

Finally, we tested the relationship between average minutes napped and CV risk factors when non-nappers (adolescents who napped 0 minutes by actigraphy or diary) were excluded from analyses. For the 10 tests conducted using the fully adjusted models, two trended. More diary-reported average minutes napped predicted lower hs-CRP ( $b = -.09$ ,  $SE = .05$ ,  $p = .075$ ) and lower HOMA-IR ( $b = .03$ ,  $SE = .02$ ,  $p = .069$ ).

## 11.0 DISCUSSION

This study utilized actigraphy and daily diary measures of daytime napping to determine whether napping was associated with elevated CV risk factors in healthy black and white adolescents. In this sample of adolescents who slept on average significantly less than the recommended nine hours per night, results were mixed, similar to the previously discussed adult literature on napping and physical health outcomes. In support of our hypothesis, more days napped by actigraphy were related to elevated IL-6. Contrary to hypothesis, more days napped by actigraphy were associated with *lower* 24-hour average SBP. Given that adolescents in this sample obtained so little sleep at night, it is possible that any additional sleep, even during the daytime, was protective for this CV risk factor. This is particularly relevant, since short nocturnal sleep is associated with elevated 24-hour SBP in this sample (Mezick, Hall, & Matthews, 2012). However, additional daytime sleep was *not* protective for IL-6, likely due to diurnal variation in IL-6, which will later be discussed in greater detail. Also against expectation, napping (by actigraphy or diary) was not associated with waist circumference, HOMA-IR, or hs-CRP. Overall, there is weak evidence that napping is related to elevated CV risk factors.

Conflictual life events moderated the relationship between actigraphy-assessed minutes napped and 24-hour SBP, but against expectation, SBP was *lower* for adolescents who reported more conflictual life events. There appears to be a protective effect of napping for adolescents who reported increased life events conflict, even after adjusting for depressive symptoms.

Considering the evidence that short nocturnal sleep is associated with poorer emotion regulation (e.g., Franzen et al., 2008), it appears that adolescents who were more vulnerable to difficulties with regulating emotion may have used napping as a coping mechanism when faced with interpersonal conflict; thus the protective effect of napping on 24-hour average SBP was stronger in this group, similar to the results found by Brindle and Conklin (2012).

Indeed, the evidence is weak that napping is associated with worse CV health. Yet this project presented a number of unexpected findings and patterns of results, which might provide fertile ground for new questions about the relationship between napping and CV risk factors in healthy adolescents, or other populations that obtain less-than-recommended amounts of nocturnal sleep. **First, we found that actigraphy, but not diary, measures of napping were associated with CV risk factors.** Although there was high correlation between actigraphy and diary measures of napping, *and* both measures were bidirectionally related to short nocturnal sleep (Jakubowski et al., 2014), these methods likely obtain somewhat different information about napping. For example, adolescents may have reported naps in their diaries only on days when they perceived feeling more tired or less rested than normal. Daily diary naps may also represent “purposeful” nap behavior, such as “escaping”/managing emotions, or perhaps “planned” or preventive naps taken prior to anticipated short nocturnal sleep (e.g., staying up late doing homework, after-school employment). In contrast, actigraphy might provide a broader range of nap behavior, capturing not only planned naps, but also “unplanned” naps that may have occurred due to excessive feelings of fatigue or sleepiness.

Additionally, since napping is a behavior that typically occurs with less regularity than nocturnal sleep, it might be more a more difficult behavior for individuals to track. When measuring naps using daily diaries, it may be worthwhile to provide more specific categories

(i.e., *Do you nap at least 30 minutes a day?*), in order to make recall of these behaviors more accessible for respondents. However, future studies should use daily diaries to collect qualitative data about nap behavior that cannot be measured using actigraphy, but could be associated with health outcomes; for example, *Did you feel refreshed after napping?*

**Second, we found that proportion of days napped predicted CV risk factors, but not average minutes napped across the study period.** The proportion of days napped variable conceptually reflects napping regularity. Comparatively, the variable for average minutes napped is a less specific construct, as adolescents could achieve the same value for average minutes napped across the study period through very different ways (i.e., taking one long nap during the week versus taking frequent shorter naps). Thus more days with a nap may reflect a worse overall sleep profile, such as shorter sleep duration and/or poorer perceived sleep quality, which may be more strongly related to CV health. Future analyses in this sample will study associations between different typologies of nocturnal sleep/napping characteristics and CV health.

**Third, napping predicted IL-6, but only after adjustment for BMI.** Vygonzas and colleagues (1997) reported a strong positive correlation between BMI and IL-6 in adults with excessive daytime sleepiness and apnea symptoms, and suggested that IL-6 was related to greater fatigue and sleepiness in these obese subjects. Thus it is possible that IL-6 is a biomarker of sleepiness in adolescents with higher BMI percentiles. However, in the present sample, BMI percentile did not moderate the relationship between daytime napping and IL-6 values (data not shown), although apnea symptoms were not explicitly measured and it is unclear whether they influenced results. It is also important to consider that IL-6 comes from sources other than adiposity, such as endothelium, myocytes and immune cells, which may suggest that people who nap have other immune differences that could not be measured in the present study. Finding



ways to improve and elongate nocturnal sleep in obese adolescents might not only reduce daytime sleepiness, but also IL-6 values.

**Fourth, although our CV risk outcomes included two measures of inflammation, napping predicted IL-6 but not hs-CRP.** As previously suggested, research suggests that IL-6 is a “sleep factor” and its serum concentration is subject to diurnal variation (Vygontzas et al., 2005; Vygontzas et al., 1997). In contrast, CRP levels lack circadian variation and generally demonstrate more stability in disease-free individuals (Meier-Ewert et al., 2001). Vygontzas and colleagues (2005) have suggested that IL-6 is a marker of sleep loss and sleepiness, which may explain why IL-6 was associated with napping but not with nocturnal sleep in this sample. In contrast, CRP has been associated with short sleep in this sample (Hall, Lee, & Matthews, 2014), but *not* with napping. It is also important to note that IL-6 plays a role in communicating with the central nervous system (CNS) to result in “sickness behaviors” that include sleep disruption and fatigue (Cho et al., 2012). In contrast, CRP does not cross the blood brain barrier nor does it influence CNS processes (except following brain injury). Thus IL-6 and CRP are very different markers of inflammation, which might further explain their different relationships with napping.

**Fifth, neither physical activity nor sedentary behaviors emerged as moderators of the napping – CV risk relationship.** Nappers (by actigraphy or diary) did not differ from non-nappers with regard to their report of physical activity or sedentary behaviors, and neither health behavior was correlated with napping variables. Although physical activity and sedentary behaviors independently predict CV risk factors (Eisenmann et al., 2008), it may be more meaningful to look at them together as an overall “activity” construct, perhaps using a ratio of engagement in physical activity versus sedentary behaviors, or creating different typologies of

physical activity/sedentary behaviors to predict CV risk. Future studies might also investigate the moderating effect of other health behaviors that are more conceptually related to sleep and sleepiness, such as caffeine use.

How do we interpret these mixed findings? Perhaps napping is not a negative health behavior for adolescents. For adolescents who obtain short sleep, increasing total sleep time by napping might be beneficial. However, this study did not take into account the *timing* of naps. Considering the two-process model of sleep, napping later in the day might further worsen nocturnal sleep and demonstrate a different relationship with CV risk factors than napping early in the day. There may also be sub-groups of adolescents for whom napping is related to poorer CV health. As seen in the present study, adolescents with a higher BMI percentile demonstrated elevated IL-6. Thus napping may be associated with worse outcomes for overweight or obese adolescents, but further research is needed to understand this relationship. Thus it is premature to state unequivocally that napping has *no* adverse consequences on adolescent CV health.

The present study has several limitations, including the use of average measures of sleep and napping, and CV risk outcomes measured at only one time point. Since actigraphy involves measurement of accelerations (Van Wouwe, Valk, Veenstra, 2011) we cannot be certain that some periods recorded as “sleep” were not actually very still moments in wake (e.g., watching TV); however our findings are strengthened by high correlation between actigraphy and diary estimates of napping. This study also had notable strengths, including multi-modal assessment of nocturnal sleep and napping, relatively even distributions of race and gender, and multiple measures of physical activity and conflict. We also had a range of CV risk outcomes, including two inflammatory markers, which have not been well-studied in relation to sleep in adolescents.

To our knowledge, the present study is the first to investigate the relationship between daytime napping and CV risk factors in healthy black and white adolescents. Overall, results provided weak evidence to suggest napping is related to worse CV risk factors. Napping may be related to elevated IL-6, but only for heavier adolescents. In contrast, napping may serve as an important mechanism for reducing systolic blood pressure, particularly in the context of high conflictual life events. In conclusion, napping may be an important factor to monitor in order to better understand the relationship between short sleep and cardiovascular health in adolescents.

## BIBLIOGRAPHY

- Acebo, C., Sadeh, A., Seifer, R., Tzischinsky, O., Wolfson, A. R., Hafer, A., & Carskadon, M. A. (1999). Estimating sleep patterns with activity monitoring in children and adolescents: How many nights are necessary for reliable measures? *Sleep*, 22, 95-103.
- Blake, G. J., & Ridker, P. M. (2002). Inflammatory bio-markers and cardiovascular risk prediction. *Journal of Internal Medicine*, 252, 283-294.
- Bondar, R. J., & Mead, D. C. (1974). Evaluation of glucose-6-phosphate dehydrogenase from *Leuconostoc mesenteroides* in the hexokinase method for determining glucose in serum. *Clinical Chemistry*, 20, 586-590.
- Borbély, A. A. (1982) A two-process model of sleep regulation. *Human Neurobiology*, 1, 195-204.
- Brindle, R. C., & Conklin, S. M. (2012). Daytime sleep accelerates cardiovascular recovery after psychological stress, *International Journal of Behavioral Medicine*, 19, 111-114.
- Buchan, D. S., Young, J. D., Boddy, L. M., Malina, R. M., & Baker, J. S. (2013). Fitness and adiposity are independently associated with cardiometabolic risk in youth. *BioMed Research International*. Advance online publication. <http://dx.doi.org/10.1155/2013/261698>.
- Campos, H., & Siles, X. (2000). Siesta and the risk of coronary heart disease: results from a population-based, case-control study in Costa Rica. *International Journal of Epidemiology*, 29, 429-437.
- Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *European Heart Journal*, 32, 1484-1492.
- Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Quantity and quality of sleep and incidence of type 2 diabetes. *Diabetes Care*, 33, 414-420.
- Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep*, 33, 585-592.

- Cappuccio, F. P., Taggart, F. M., Kandala, N. B., Currie, A., Peile, E., Stranges, S., & Miller, M. A. (2008). Meta-analysis of short sleep duration and obesity in children and adults. *Sleep*, *31*, 619-626.
- Centers for Disease Control and Prevention. 2007 Youth Risk Behavior Survey. 2007. [www.cdc.gov/yrbss](http://www.cdc.gov/yrbss). Accessed 7/12/13.
- Chen, X., Beydoun, M. A., & Wang, Y. (2008). Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity*, *16*, 265-274.
- Cho, H. J., Bower, J. E., Kiefe, C. I., Seeman, T. E., & Irwin, M. R. (2012). Early life stress and inflammatory mechanisms of fatigue in the coronary artery risk development in young adults (CARDIA) study. *Brain, Behavior, and Immunity*, *26*, 859-865.
- Countryman, A. J., Saab, P. G., Llabre, M. M., Penedo, F. J., McCalla, J. R., & Schneiderman, N. (2013). Cardiometabolic risk in adolescents: Associations with physical activity, fitness, and sleep. *Annals of Behavioral Medicine*, *45*, 121-131.
- Danesh, J., Whincup, P., Walker, M., Lennon, L., Thomson, A., Appleby, P., ... Pepys, M. B. (2000). Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. *British Journal of Medicine*, *321*, 199-204.
- Eisenmann, J. C., Barteel, R. T., Smith, D. T., Welk, G. J., & Fu, Q. (2008). Combined influence of physical activity and television viewing on the risk of overweight in US youth. *International Journal of Obesity*, *32*, 613-618.
- de Ferranti, S., & Rifai, N. (2002). C-reactive protein, inflammation, and coronary risk. *Cardiology Clinics*, *21*, 315-325.
- Fischer, F. M., Nagai, R., Teixeira, L. R. (2008). Explaining sleep duration in adolescents: the impact of socio-demographic and lifestyle factors and working status. *Chronobiology International*, *25*, 359-372.
- Flint, J., Kothare, S. V., Mamoon, Z., Suarez, E., Adams, R., Legido, A., & De Luca, F. (2007). Association between inadequate sleep and insulin resistance in obese children. *Journal of Pediatrics*, *150*, 364-369.
- Franzen, P. L., Buysse, D. J., Dahl, R. E., Thompson, W., & Siegle, G. J. (2009). Sleep deprivation alters papillary reactivity to emotional stimuli in healthy young adults. *Biological Psychology*, *80*, 300-305.
- Franzen, P. L., Siegle, G. J., & Buysse, D. J. (2008). Relationships between affect, vigilance, and sleepiness following sleep deprivation. *Journal of Sleep Research*, *17*, 34-41.
- Grandner, M. A., Buxton, O. M., Jackson, N., Sands-Lincoln, M., Pandey, A., Jean-Louis, G.

- (2013). Extreme sleep durations and increased C-reactive protein: effects of sex and ethnorracial group. *Sleep*, 36, 769-799E.
- Gallicchio, L., & Kalesan, B. (2009). Sleep duration and mortality: a systematic review and meta-analysis. *Journal of Sleep Research*, 18, 148-158.
- Garaulet, M., Ortega, F. B., Ruiz, J. R., Rey-Lopez, J. P., Beghin, L., Manios, Y., ... Moreno, L. A. (2011). Short sleep duration is associated with increased obesity markers in European adolescents: effect of physical activity and dietary habits. The HELENA study. *International Journal of Obesity*, 35, 1308-1317.
- Guo, X., Zheng, L., Wang, J., Zhang, X., Zhang, X., Li, J., & Sun, Y. (2013). Epidemiological evidence for the link between sleep duration and high blood pressure: A systematic review and meta-analysis. *Sleep Medicine*, 14, 324-332.
- Hall, M. H., Lee, L., & Matthews, K. A. (in press). Sleep duration during the school week is associated with C-reactive protein risk groups in healthy adolescents. *Sleep Medicine*.
- Hamilton, M. T., Healy, G. N., Dunstan, D. W., Zderiz, T. W., & Owen, N. (2008). Too little exercise and too much sitting: inactivity physiology and the need for new recommendations on sedentary behavior. *Current Cardiovascular Risk Reports*, 2, 292-298.
- Hartiala, O., Magnussen, C. G., Kajander, S., Knuuti, J., Ukkonen, H., Saraste, A., ... Raitakari, O. T. (2012). Adolescence risk factors are predictive of coronary artery calcification at middle age: The Cardiovascular Risk in Young Finns Study. *Journal of the American College of Cardiology*, 60, 1364-1370.
- Hollingshead, A. B. (1975). Four factor index of social status. New Haven, CT: Yale University; 1975.
- Jakubowski, K. P., Hall, M. H., Lee, L., & Matthews, K. A. (2014). *Napping in healthy black and white adolescents*. Manuscript under review.
- Javaheri, S., Storfer-Isser, A., Rosen, C. L., & Redline, S. (2008). Sleep quality and elevated blood pressure in adolescents. *Circulation*, 118, 1034-1040.
- Javaheri, S., Storfer-Isser, A., Rosen, C. L., & Redline, S. (2011). Association of short and long sleep durations with insulin sensitivity in adolescents. *Journal of Pediatrics*, 158, 617-623.
- Koren, D., Levitt Katz, L. E., Brar, P. C., Gallagher, P. R., Berkowitz, R. I., & Brooks, L. J. (2011). Sleep architecture and glucose and insulin homeostasis in obese adolescents. *Diabetes Care*, 34, 2442-2447.
- Kushida C. A., Chang A., Gadkary C., Guilleminault C., Carrillo O., & Dement WC. (2001)

- Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep-disordered patients. *Sleep Medicine*, 2, 389–396.
- Leng, Y., Wainwright, N. W. J., Cappuccio, F. P., Surtees, P. G., Hayat, S., Luben, R., ... Khaw, K-T. (2014). Daytime napping and the risk of all-cause and cause-specific mortality: a 13-year follow-up of a British population. *American Journal of Epidemiology*. Advance online publication. doi. 10.1093/aje/kwu036
- Li, S., Chen, W., Srinivasan, S. R., Bond, M. G., Tang, R., Urbina, E. M., & Berenson, G. S. (2003). Childhood cardiovascular risk factors and carotid vascular changes in adulthood: The Bogalusa Heart Study. *JAMA*, 290, 2271-2276.
- Li, C., Ford, E. S., Zhao, G., & Mokdad, A. H. (2009). Prevalence of pre-diabetes and its association with clustering of cardiometabolic risk factors and hyperinsulinemia among U.S. adolescents: National Health and Nutrition Examination Survey 2005-2006. *Diabetes Care*, 32, 342-347.
- Martinez-Gomez, D., Eisenmann, J. C., Gomez-Martinez, S., Hill, E. E., Zapatera, B., Veiga, O. L., & Marcos, A. (2011). Sleep duration and emerging cardiometabolic risk markers in adolescents. The AFINOS study. *Sleep Medicine*, 12, 997-1002.
- Masten, A., Neeman, J., & Andenas, S. (1994). Life events and adjustment in adolescents: The significance of event independence, desirability, and chronicity. *Journal of Research on Adolescence*, 4, 71- 97.
- Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: insulin resistance and beta-cell functioning from fasting plasma glucose and insulin concentrations in man. *Diabetologia*, 28, 412-419.
- Matthews, K. A., Dahl, R. E., Owens, J. F., Lee, L., & Hall, M. (2012). Sleep duration and insulin resistance in healthy black and white adolescents. *Sleep*, 35, 1353-1358.
- Matthews, K. A., Zheng, H., Kravitz, H., Sowers, M., Bromberger, J. T., Buysse, D. J., ... Hall, M. (2010). Are inflammatory and coagulation biomarkers related to sleep characteristics in mid-life women?: Study of Women's Health Across the Nation Sleep Study. *Sleep*, 33, 1649-1655.
- McKnight-Eily, L. R., Eaton, D. K., Lowry, R., Croft, J. B., Presley-Cantrell, L., & Perry, G. S. (2011). Relationships between hours of sleep and health-risk behaviors in US adolescent students. *Preventive Medicine*, 53, 271-273.
- Meier-Ewert, H. K., Ridker, P. M., Rifai, N., Price, N., Dinges, D. F., & Mullington, J. M. (2001). Absence of diurnal variation of C-reactive protein concentrations in healthy human subjects. *Clinical Chemistry*, 47, 426-430.
- Meng, L., Zheng, Y., & Hui, R. (2013). The relationship of sleep duration and insomnia to risk

- of hypertension incidence: A meta-analysis of prospective cohort studies. *Hypertension Research*, 36, 985-995.
- Mezick, E. J., Hall, M. & Matthews, K. A. (2012). Sleep duration and ambulatory blood pressure in black and white adolescents. *Hypertension*, 59, 747-752.
- Miller, M. A., Kandala, N-B., Kivimaki, M., Kumari, M., Brunner, E. J., Lowe, G. D. O., ... Cappuccio, F. P. (2009). Gender differences in the cross-sectional relationships between sleep duration and markers of inflammation: Whitehall II study. *Sleep*, 32, 857-864.
- Muntner, P., He, J., Cutler, J. A., Wildman, R. P., & Whelton, P. K. (2004). Trends in blood pressure among children and adolescents. *JAMA*, 291, 2107-2113.
- Naska, A., Oikonomou, E., Trichopoulou, A., Psaltopoulou, T., & Trichopoulos, D. (2007). Siesta in healthy adults and coronary mortality in the general population. *Archives of Internal Medicine*, 167, 296-301.
- National Sleep Foundation. Teens and Sleep. 2006.  
[http://www.sleepfoundation.org/sites/default/files/2006\\_summary\\_of\\_findings.pdf](http://www.sleepfoundation.org/sites/default/files/2006_summary_of_findings.pdf).  
 Accessed 11/5/13.
- National Sleep Foundation. Communications Technology in the Bedroom. 2011.  
[http://www.sleepfoundation.org/sites/default/files/sleepinamericapoll/SIAP\\_2011\\_Summary\\_of\\_Findings.pdf](http://www.sleepfoundation.org/sites/default/files/sleepinamericapoll/SIAP_2011_Summary_of_Findings.pdf). Accessed 11/5/13.
- Nelson, M. C., & Gordon-Larsen, P. (2006). Physical activity and sedentary behavior patterns are associated with selected adolescent health risk behaviors. *Pediatrics*, 117, 1281-1290.
- Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA*, 303, 242-249.
- Palagini, L., Bruno, R. M., Gemignani, A., Baglioni, C., Ghiadoni, L., & Riemann, D. (2013). Sleep loss and hypertension: A systematic review. *Current Pharmaceutical Design*, 19, 2409-2419.
- Patel, S. R. (2007). Is siesta more beneficial than nocturnal sleep? *Archives of Internal Medicine*, 167, 2143-2144.
- Patel, S. R., & Hu, F. B. (2008). Short sleep duration and weight gain: a systematic review. *Obesity*, 16, 643-653.
- Peterson, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17, 117-133.
- Platat, C., Wagner, A., Klumpp, T., Schweitzer, B., & Simon, C. (2006). Relationships of



- physical activity with metabolic syndrome features and low-grade inflammation in adolescents. *Diabetologia*, 49, 2078-2085.
- Rademacher, E. R., Jacobs, D. R., Jr., Moran, A., Steinberger, J., Prineas, R. J., & Sinaiko, A. (2009). Relation of blood pressure and body mass index during childhood to cardiovascular risk factor levels in young adults. *Journal of Hypertension*, 27, 1766-1774.
- Radloff, L. S. (1977). The CES-D scale: a self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385-401.
- Raitakari, O. T., Juonala, M., Kähönen, M., Taittonen, L., Laitinen, T., Mäki-Torkko, N., ... Viikari, J. S. A. (2003). Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: The Cardiovascular Risk in Young Finns Study. *JAMA*, 290, 2277-2283.
- Roberts, R. E., Roberts, C. R., & Duong, H. T. (2009). Sleepless in adolescence: prospective data on sleep deprivation, health and functioning. *Journal of Adolescence*, 32, 1045-1057.
- Roberts, R. E., Roberts, C. R., & Xing Y. (2011). Restricted sleep among adolescents: prevalence, incidence, persistence, and associated factors. *Behavioral Sleep Medicine*, 9, 18-30.
- Spruyt, K., Molfese, D. L., & Gozal, D. (2011). Sleep duration, sleep regularity, body weight, and metabolic homeostasis in school-aged children. *Pediatrics*, 127, e345-e352.
- Stang, A., Dragano, N., Moebus, S., Möhlenkamp, S., Schmerund, A., Kalsch, H., ... Jöckel, K. H. (2012). Midday naps and the risk of coronary artery disease: results of the Heinz Nixdorf Recall Study. *Sleep*, 35, 1705-1712.
- Stea, T. H., Knutsen, T., & Torstveit. (2014). Association between short time in bed, health-risk behaviors and poor academic achievement among Norwegian adolescents. *Sleep Medicine*, 15, 666-671.
- Stone, K. L., Ewing, S. K., Ancoli-Israel, S., Ensrud, K. E., Redline, S., Bauer, D. C., ... Cummings, S. R. (2009). Self-reported sleep and nap habits and risk of mortality in a large cohort of older women. *Journal of the American Geriatric Society*, 57, 604-611.
- Taheri, S., Austin, D., Lin, L., Nieto, F. J., Young, T., & Mignot, E. (2007). Correlates of serum C-reactive protein (CRP) – No association with sleep duration or sleep disordered breathing. *Sleep*, 30, 991-996.
- Tanabe, N., Iso, H., Seki, N., Suzuki, H., Yatsuya, H., Toyoshima, H., & Tamakoshi, A., & JACC Study Group. (2010). Daytime napping and mortality, with a special reference to cardiovascular disease: the JACC study. *International Journal of Epidemiology*, 39, 233-243.

- Tremblay, M. S., LeBlanc, A. G., Kho, M. E., Saunders, T. J., Larouche, R., Colley, R. C., ... Gorber, S. C. (2011). Systematic review of sedentary behavior and health indicators in school-aged children and youth. *International Journal of Behavioral Nutrition and Physical Activity*, 8, 98. doi:10.1186/1479-5868-8-98.
- Tryon, W. W. (2004). Issues of validity in actigraphic sleep assessment. *Sleep*, 27, 158–165.
- Van Eyck, A., Van Hoorenbeeck, K., De Winter, B. Y., Ramet, J., Van Gaal, L., De Backer, W., & Verhulst, S. L. (2013). Sleep-disordered breathing and C-reactive protein in obese children and adolescents. *Sleep and Breathing*, 18, 335-340.
- Van Wouwe, N. C., Valk, P. J. L., & Veenstra, B. J. (2011). Sleep monitoring: a comparison between three wearable instruments. *Military Medicine*, 176, 811-816.
- Vygontzas, A. N., Bixler, E. O., Lin, H.-M., Prolo, P., Trakada, G., & Chrousos, G. P. (2005). IL-6 and its circadian secretion in humans. *Neuroimmunomodulation*, 12, 131-140.
- Vygontzas, A. N., Papanicolaou, D. A., Bixler, E. O., Kales, A., Tyson, K., & Chrousos, G. P. (1997). Elevation of plasma cytokines in disorders of excessive daytime sleepiness: role of sleep disturbance and obesity. *Journal of Clinical Endocrinology and Metabolism*, 82, 1313-1316.
- Wang, Q., Xi, B., Liu, M., Zhang, Y., & Fu, M. (2012). Short sleep duration is associated with hypertension risk among adults: A systematic review and meta-analysis. *Hypertension Research*, 35, 1012-1018.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, 54, 1063-1070.
- Wolfson, A., & Carskadon, M. A. (1998). Sleep schedules and daytime functioning in adolescents. *Child Development*, 69, 875-887.
- Xi, B., He, D., Zhang, M., Xue, J., & Zhou, D. (2013). Short sleep duration predicts risk of metabolic syndrome: A systematic review and meta-analysis. *Sleep Medicine Reviews*, 18, 293-297.
- Yoo, S-S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep: a pre-frontal-amygdala disconnect. *Current Biology*, 17, 877-878.